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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-seventh Annual Meeting

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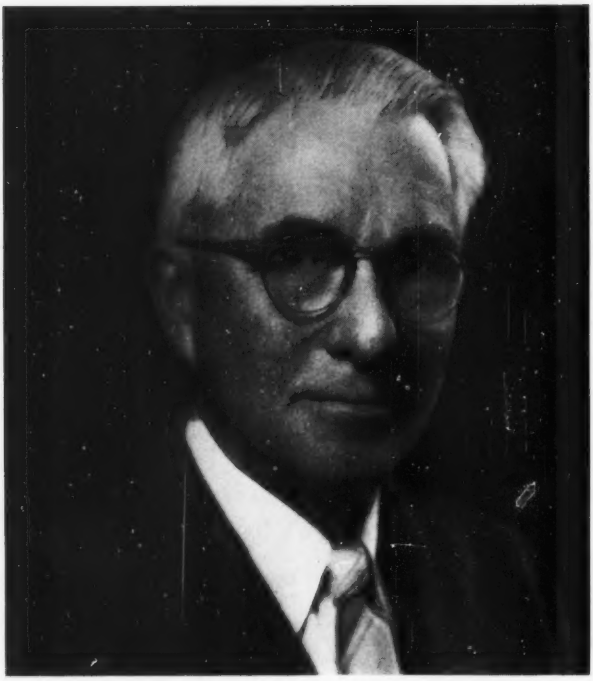
September 24, 25 and 26, 1953

Cleveland, Ohio



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HAROLD JUDSON LEONARD, D.D.S.

Founder and Secretary-Treasurer since its inception of the American Board of Periodontology

Harold Judson Leonard was born in Minneapolis, Minnesota and received his Doctor of Dental Surgery degree from the University of Minnesota in 1912 and his B.A. in 1915. He has excelled as teacher, research worker, writer and practitioner in periodontology and oral diagnosis; is a Fellow and past President, American Academy of Periodontology; Fellow, American Association for the Advancement of Science; founder and Secretary since its inception to 1951 of the Advisory Board of Dental Specialties, among other responsibilities and honors from organized dentistry.

Dr. Leonard is married, has four sons and seven grandchildren, practices in New York City and lives in Scarsdale, N. Y. He reports he has some time left for his hobbies, gardening and Boy Scout work.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-THREE

The Effects of Various Levels of Dietary Protein on the Periodontal Tissues of Young Rats

by ASGER M. FRANDSEN, D.D.S.; HERMANN BECKS, D.D.S.; MARJORIE M. NELSON, PH.D.; HERBERT M. EVANS, M.D., *San Franciso and Berkeley, California*

RELATIVELY few data are available concerning a possible relationship of experimental protein deficiencies and oral pathology. The effects of protein deprivation in the rat have been described as diminished alveolar bone formation, reduced number of trabeculae in the supporting bone and decreased amount of collagen fibres in the periodontal membrane.[1] Root and alveolar bone resorption[7] and traumatic lesions of the interdental papilla have also been reported.[5]

In a previous paper[3] the effects of various levels of protein deprivation on skeletal growth and endochondal ossification were described. The bone changes in the proximal end of the tibia consisted of decreased width of the epiphyseal disc with an increase in the relative amount of cartilage ground substance and decreased activity in the zone of erosion resulting in only a few coarse bone trabeculae. Growth in length of the tibia was severely retarded.

The present report deals with the changes seen in the periodontal tissues of the animals used in the above mentioned investigation.[3]

MATERIAL AND EXPERIMENTAL ARRANGEMENT

Forty-six male rats of the Long-Evans strain were weaned when 21 days old and divided into seven groups (Table I). Four purified diets were used, varying in the casein content (Table II). The control diet (A) contained 24% casein, whereas the deficient diets (B, C and D) contained 6%, 3% and 0% casein respectively. The casein deficit was compensated for by the same amount of sucrose, thus making the diets isocaloric. The food intake was recorded daily,

From the Division of Dental Medicine, College of Dentistry, the George Williams Hooper Foundation for Medical Research, and the Institute of Experimental Biology, University of California, San Francisco, and Berkeley.

Aided by grants from the American Foundation for Dental Science, the Board of Research and the Department of Agriculture of the University of California, the Roche Anniversary Foundation, and the California State Dental Association. The following materials were generously contributed: crystalline B vitamins and alphatocopherol from Merck and Company, Inc., Rahway, New Jersey; liver powder (lab. no. 7-5293) from Lederle Laboratories, Inc., Pearl River, New York.

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TABLE I

Experimental Arrangement

Group	*No. of Rats*	*Diet*	*Age at Autopsy*
Ad lib control	12	Diet A (24% casein)	63-67 days
Protein deficient	6	Diet B (6% casein)	63 days
Pairfed control	6	Diet A	63 days
Protein deficient	6	Diet C (3% casein)	67 days
Pairfed control	6	Diet A*	67 days
Protein deficient	5	Diet D (0% casein)	67 days
Pairfed control	5	Diet A*	67 days

*See Table II for vitamin supplementation of these diets.

and the rats were weighed every fifth day. They had free access to drinking water (distilled) and were kept in screen bottom cages.

The animals were autopsied between 63 and 67 days of age after approximately six weeks on the diets. The length of the experimental periods was determined by the moribund condition of the protein deficient animals. The heads were fixed in 10% neutral formol, cut sagittally in the midline and each half roentgenographed. The mandibles were dissected and the right halves were decalcified in 5% nitric acid, embedded in nitrocellulose, and sectioned mesio-distally and stained with hematoxylin and eosin.

RESULTS

A. Roentgenographic Findings.

The size of the skulls is indicated by reproductions of roentgenograms representative of the groups on the purified control diet ad lib (Fig. 1), the 0% casein diet (Fig. 2), and its pairfed control (Fig. 3). In addition the radiolucency of the skull and the alveolar process of the protein-free group may be noted.

B. Histologic Findings.

Survey photomicrographs of the periodontal tissues of the second and third mandibular molars are seen in Figs. 4-6. Fig. 4 represents an ad libitum control receiving the 24% casein diet. The interdental papilla

TABLE II

Diets

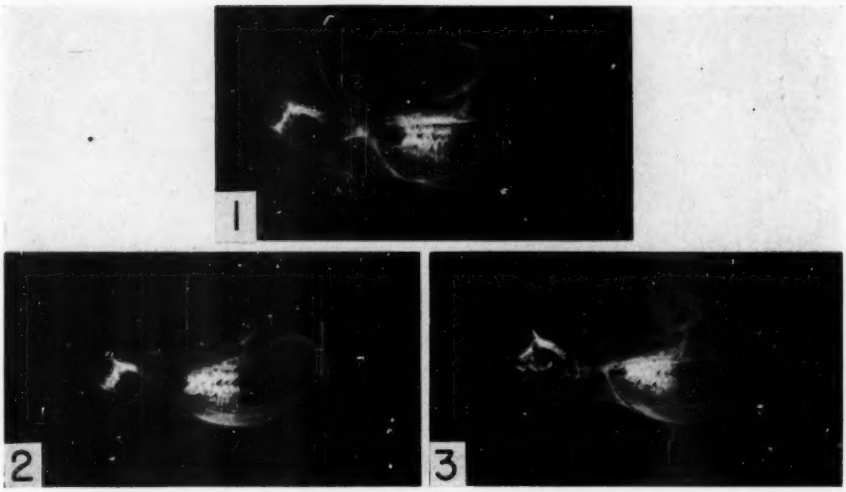
	Diet A %	*Diet B* %	*Diet C* %	*Diet D* %
Alcohol-extracted casein	24	6	3	0
Sucrose	63	81	83	86
Hydrogenated vegetable oil	8	8	8	8
Salts No. 4 (4)	4	4	4	4
Liver Powder (a)	0.5	0.5	1.0*	1.0*
B. Vitamin Mixture (b)	0.5	0.5	1.0*	1.0*

(a) 1 gram of Liver Fraction powder (Lederle La., No. 7-5293) furnished 550 μg. L. casei factor (folic acid) and at least 5 μg. B_{12}.

(b) 100 grams of diet contained 30 μg. biotin, 275 μg. pteroylglutamic acid, 500 μg. thiamine HCL, 500 μg. pyridoxine HCL, 1mg. riboflavin, 500 μg. 2-methyl-1, 4, naphthoquinone, 1 mg. p-aminobenzoic acid, 2 mg. nicotinic acid, 5 mg. calcium pantothenate, 40 mg. inositol, and 100 mg. choline chloride.

All animals received weekly 1 cc. of a fat-soluble vitamin mixture which furnished 6 mg. alpha-tocopherol (Merck), 115 chick units vitamin D, 800 U.S.P. units vitamin A, and 650 mg. corn oil (Mazola).

*In diets C and D and in their corresponding pairfed control diets these vitamin levels were doubled.

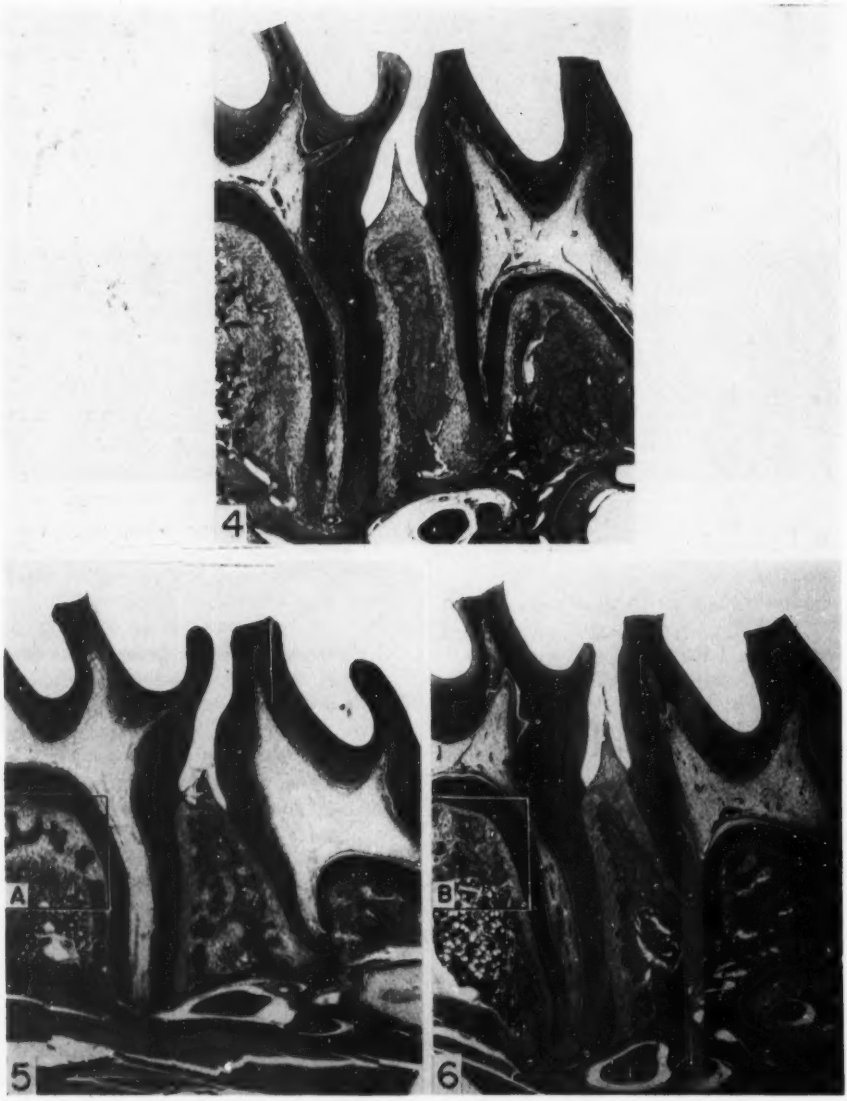


Figs. 1-3. Roentgenograms of right half of head. Orig. Magnif. × 1.8.
Fig. 1. Ad libitum control group. 67 days old. Fig. 2. 0% casein group. 67 days old. Fig. 3. Pair-fed with 0% casein group. 67 days old.

is intact and the periodontal membrane is rich in cells and well defined collagen fibers. The cementum consists of a distinct, heavy basophilic layer. Around the apices a substantial formation of osteocementum is noted. The alveolar bone consists of an uninterrupted lamina compacta showing the resorption-apposition pattern indicative of the physiologic distal migration of the molars;[6] the distal surface of the interdental septum is covered with a bead-like arrangement of osteoblasts whereas the mesial surface shows vacuolization indicating resorption. Two areas of resorption are noted on the distal surface of the second molar root. The supporting bone is dense, showing few, small marrow cavities. The sparse amounts of marrow tissue appear to be of hematopoietic type.

The group given a 0% diet casein is represented by the specimen in Fig. 5. The root, as well as the vertical dimension of the mandible, are shorter. The pulp chambers and canals are relatively large with only a faint differentiation of odontoblasts. The interdental papilla has almost disappeared and the periodontal membrane shows few cells and indistinct principal fibers. The third molar impinges on the alveolar bone in some areas of the bifurcation. The cementum of the third molar and in the bifurcation of the second is thin, and the formation of osteocementum negligible. The alveolar bone has lost its character of a lamina compacta, and of the remodelling activities only resorption is in evidence. There is a definite osteoporosis of the supporting bone with some fibrous displacement of the marrow tissue.

The pairfed control (Fig. 6) shows larger dimensions of teeth and jaw. The pulp chamber and canals are relatively small with a well-differentiated layer of odontoblasts. The interdental papilla is intact and the principal fibers of the periodontal membrane are clearly indicated. There is a distinct layer of cementum and a considerable formation of osteocementum on the second molar. The alveolar bone consists of an almost uninterrupted lamina compacta. The interdental septum shows evidence of resorption on the mesial surface, whereas



Figs. 4-6. Survey photomicrographs of lower right second and third molars. Orig. Magnif. × 40. Fig. 4. Ad libitum control group. 67 days old. Fig. 5. 0% casein group. 67 days old. Fig. 6. Pair-fed with 0% casein group. 67 days old.

no apposition on the distal surface is apparent. The supporting bone contains rather large marrow cavities with fatty infiltration of the hematopoietic tissue.

The areas designated as A and B in the

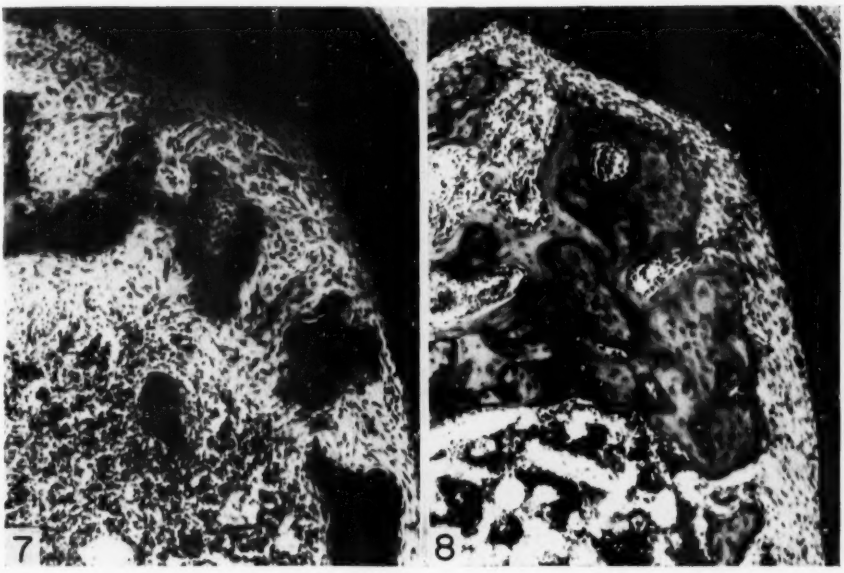


Fig. 7. Higher magnification of area A from Fig. 5. Orig. Magnif. × 190. Fig. 8. Higher magnification of area B from Fig. 6. Orig. Magnif. × 190.

survey photomicrographs are shown at a higher magnification in Figs. 7 and 8. In the specimen from the 0% casein group (Fig. 7) the pronounced destruction of the alveolar bone is noted. Only a few fragments of bone persist and resorptive activities are evident. There is practically no supporting bone present and fibrous tissue is invading the marrow cavity. The pairfed control animal (Fig. 8) exhibits a fairly well developed bony structure. Newly formed bone is present, but the cells lining the surface of the trabeculae are small and flattened indicating that no bone formation was taking place at the time of sacrifice.

The interdental papillae and the alveolar crests from the various groups are shown in Figs. 9-15. Fig. 9 illustrates an ad libitum fed control animal. The epithelium is intact and is, for the main part, only a few cell-layers thick. The connective tissue body of the papilla consists of loosely arranged, vascular fibrous tissue with essentially no cellular infiltration. The transseptal principal fibres extend from one tooth to the other in a distinct uninterrupted bundle. The alveolar crest consists of compact lamellated bone, with osteoblasts lining the distal surface, while resorption lacunae are seen on the mesial surface. In a rat from the protein-fed group (Fig. 10) the papilla has almost disintegrated. The epithelial cells are loosely arranged, and covered with inflammatory exudate. The amount of loose connective tissue is negligible leaving the epithelium resting directly on the transseptal fibres. These are indistinct and their continuity is broken. The alveolar crest is undergoing resorption on both the mesial and the distal side. The pairfed control (Fig. 13) has an intact papilla, and well established transseptal fibres. The alveolar crest shows resorption on the mesial side, but neither resorption nor apposition on the distal. In the 3% casein group (Fig. 11) the papilla shows some disintegration and epithelial proliferation, and only a small amount of loose connective tissue between the epithelium and the transseptal fibres,

which are clearly defined. The alveolar crest is compact with active resorption of the mesial surface, while the distal appears to be mainly inactive. The pairfed control (Fig. 14) has an intact papilla and shows bone apposition on the distal side of the alveolar crest. Fig. 12 illustrates the 6% casein group, in which the papilla shows a normal outline, but has some epithelial proliferation and cellular infiltration of the lamina compacta. The transseptal fibres are readily seen, and the compact alveolar crest shows mesial resorption and distal apposition. Except for a lesser degree of epithelial proliferation, the pairfed control (Fig. 13) does not appear to show any difference from the deficient animal.

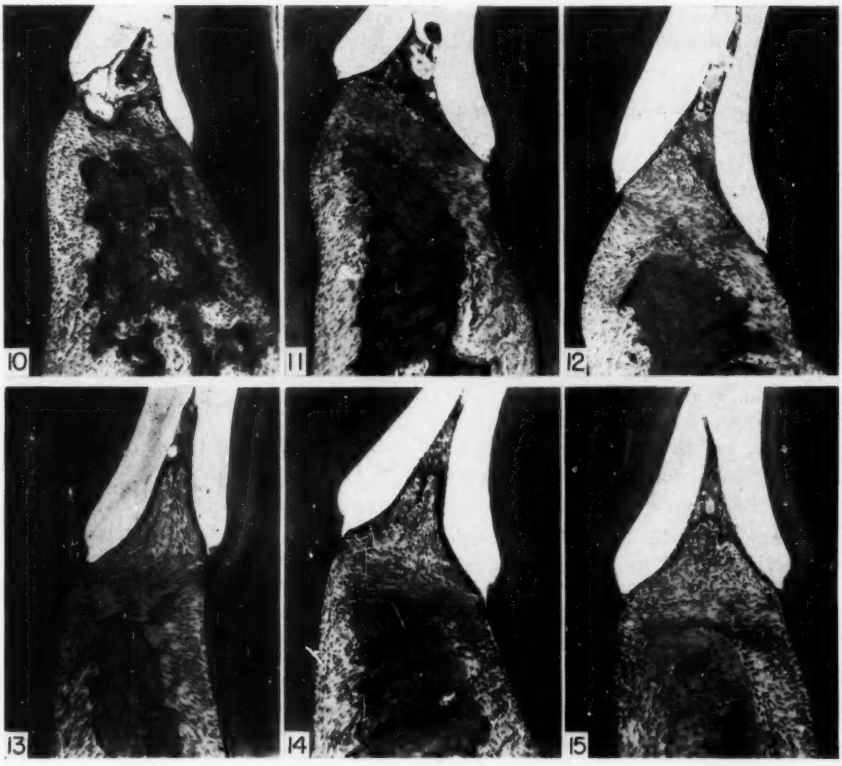
If the three levels of protein deprivation are compared (Figs. 10, 11, 12), it is noted that, whereas the diet containing 6% casein provides for an almost intact papilla with slight epithelial proliferation, the 3% casein, and especially the 0% casein diet cause a marked decrease in the connective tissue core of the papilla. Concomitantly epithelial proliferation and inflammatory exudate is increased. The transseptal fibres are maintained in the 6% and the 3% casein groups, but are absent or severely broken up in the 0% casein group. As for the alveolar crest, it appears that 6% casein is capable of maintaining the resorption-apposition pattern, while little or no apposition takes place at the 3% casein level. In the protein-free group the pronounced resorption gives the impression of an acute tissue break-down with no attempts of replacement.

The three groups of pairfed rats (Figs. 13, 14, 15) furnish material for an evaluation of the effects of severe dietary restriction of an adequate diet. On calculating the food intake of these groups, it was found that the animals pairfed with the 0% casein group received 21% of the food intake consumed by the ad libitum fed controls. For the animals pairfed with the 3% and the 6% casein group, these figures were 32% and 45% respectively. Dietary restriction to 45% and 32% (Figs. 14, 15) does not appear to cause any marked changes in the interdental tissues illustrated. A decrease in the caloric intake to 21% of the normal (Fig. 13) will prevent bone apposition on the distal side of the alveolar crest and also cause a decreased cellularity of the interdental papilla and the periodontal membrane.

Fig. 9. Ad libitum control group. 67 days old. Interdental area between lower right second and third molars. Orig. Magnif. × 165.

COMMENTS

Root and alveolar bone resorption in the molar area has been ascribed to protein deficiency.[7] It should be kept in mind that these activities are encountered in normal growing rats, especially on the distal surface of the roots and on the mesial surfaces of the alveolar bone associated with the physiologic distal migration of the molars. In our material root resorption is seen primarily in the ad libitum controls (Fig. 4) whereas it is a rare finding in the deficient animals. This may be explained by the decreased growth activities in the latter.[3]



Figs. 10-15. Interdental area between lower right second and third molars. Orig. Magnif. × 165. Fig. 10. 0% casein group. 67 days old. Fig. 11. 3% casein group. 67 days old. Fig. 12. 6% casein group. 63 days old. Fig. 13. Pairfed with 0% casein group. 67 days old. Fig. 14. Pairfed with 3% casein group. 67 days old. Fig. 15. Pairfed with 6% casein group. 63 days old.

Traumatic lesions of the interdental tissues as reported by Hunter,[5] were not seen in our material, probably because our animals were raised from weaning on a finely ground purified diet, while his received Chow for 15 weeks prior to the experiment.

The findings of Chawla and Glickman[1] with regard to the decreased amount of principal fibers, arrest of alveolar bone apposition and osteoporosis of the supporting bone were generally confirmed by this study.

Atrophy and wasting away of tissue are important findings in protein deficiencies.[3] In the periodontal tissues this is primarily expressed in the preponderance of the catabolic over the anabolic tissue changes seen in the protein free group. The fact that the alveolar bone of the bifurcation appears to be the most severely affected may be explained from a functional viewpoint. Only few principal fibres are normally present here, hence this part plays a minor role in supporting the tooth. The trabeculae of the supporting bone are not directly involved in the attachment of the

fibers, and they, likewise, disappear rather than the functional parts of the alveolar bone.

The question may be raised whether the severe osteoporosis seen in the protein free groups (Figs. 5 and 10) is due entirely to lack of bone apposition, or to a combination of this and an increased rate of resorption. Some clarification may be obtained by comparing it with the 3% casein group and the group pairfed with the protein-free animals. The casein and the dietary restriction of these two groups respectively has caused the inhibition of practically all bone apposition. By comparison, the protein-free group shows distinct osteoporosis. It is, therefore, suggested that the protein-free diet is responsible for an increased rate of bone resorption.

It may finally be added that ulceration of the interdental papilla associated with epithelial proliferation has been reported for dogs[2] and rats[8] deficient in some members of the vitamin B complex.

SUMMARY

The effects on the periodontal structures of different levels of dietary protein was studied in growing male rats. Casein, in purified diets, was given at a 6%, 3% and 0% level. The control diet contained 24% casein and was given ad libitum as well as restricted to the amount consumed by the deficient animals. The experimental period extended from the day of weaning until 63-67 days of age.

The protein deficient animals had smaller jaws and less osteocementum on the roots than the pairfed controls. They also showed interference with the integrity of the interdental papillae, and when completely deprived of protein, marked disturbance of the transseptal fibres. Osteoporosis of the alveolar and supporting bone became accentuated with increasing protein deprivation and led to severe breakdown of bone tissue in the protein-free group where one may assume an increased rate of resorption. The normal distal shifting of the molars was arrested in the 3% and 0% casein group and in the animals pairfed with the 0% group, indicated by the lack of bone apposition on the distal surface of the interdental septae.

REFERENCES

1. Chawla, T. N., Glickman, I.: Protein Deprivation and the Periodontal Structures of the Albino Rat. Oral Surgery, Oral Med., & Oral Path. **4**:578, 1951.

2. Becks, H., Wainwright, W. W., Morgan, A. F.: Comparative Study of Oral Changes in Dogs Due to Pantothenic Acid, Nicotinic Acid and Unknowns of the B Vitamin Complex. Am. J. Orthodontics and Oral Surgery **29**:183, 1943.

3. Frandsen, A. M., Nelson, M. M., Sulon, E., Becks, H., Evans, H. M.: The Effects of Various Levels of Dietary Protein on Skeletal Growth and Endochondral Ossification in Young Rats. (In preparation).

4. Hegsted, D. M., Mills, R. C., Elvehjem, C. A., Hart, E. B.: Choline in the Nutrition of the Chick. J. Biol. Chem., **138**:459, 1941.

5. Hunter, H. A.: Hypoproteinemia in Relation to the Dental Tissues. J. Dent. Res., **29**:73, 1950.

6. Sicher, H., Weinmann, J. P.: Bone Growth and Physiologic Tooth Movement. Am. J. Orthodontics, **30**:109, 1944.

7. Stein, G., Ziskin, D. E.: The Effect of a Protein-Free Diet on the Teeth and Periodontium of the Albino Rat. J. Dent. Res., **28**:529, 1949. (Abstract)

8. Wainwright, W. W., Nelson, M. M.: Changes in the Oral Mucosa Accompanying Acute Pantothenic Acid Deficiency in Young Rats. Am. J. Orthodontics and Oral Surgery, **31**:406, 1945.

Clinical Manifestation of Periodontal Disturbances Requiring Treatment

By EDGAR D. COOLIDGE, *Chicago, Illinois*

DURING the last quarter of a century there has been much discussion of the etiology of periodontal lesions, the nomenclature and classification of the various tissue changes that are to be found in the periodontal tissues. Lengthy discussions have been recorded where different opinions were at variance by only the name by which the same tissue changes might be identified. Most of these discussions and a certain percent of the literature of recent years have been concerned with an attempt to simplify and unify the prevailing concepts of periodontal diseases. While much has been accomplished there is still much confusion in the minds of many as to the present classification of the lesions.

The commonly used terms, pyorrhea and periodontoclasia, which are very general terms, have been more or less replaced by the terms periodontitis and periodontosis to indicate a difference between the inflammatory and the non-inflammatory periodontal lesions.

It is rare that periodontosis can be identified in clinical practice until the pocket develops and then it is no longer known as periodontosis, but as periodontitis complex. The long struggle to develop a concept of a degenerative disease to be found in the periodontal tissues only by microscope and very rarely by that method of examination and then to further learn that as soon as we can observe the condition it must be called by another name, periodontitis complex, leaves something still to be desired by the dentist who stands beside the patient in the dental office.

The discussion of the etiology and the pathology of a condition that is mostly academic has doubtless encouraged and stimulated much interest and has enlightened some and confused many. When one stops to consider that practically all periodontal lesions requiring treatment are of the inflammatory type and must be treated as such with much the same manner of treatment, would it not be appropriate to turn our attention to the periodontal pocket and ask ourselves "what about the pocket."

THE PERIODONTAL POCKET

Periodontal diseases, as dealt with by the clinician, are usually of an inflammatory character. The presence of local irritants including calculus, bacteria and their products or any other form of irritation to the gingival collar calls forth from the body the elements of defense which accumulate in the tissue surrounding the gingival sulcus or pocket. The gingival sulcus is the starting place in which the destructive forces from without are met by the defensive mechanism from within. The outcome of the struggle between these two forces depends upon many factors. It is more surprising that the defensive mechanism overcomes the invader so often and the gingiva is maintained in normal relationship to the teeth than that frequently the attacking forces and irritants lead to pocket formation. However it must be stated here, as it has often been stated elsewhere, that more teeth are lost because of periodontal disease than by any other cause. The clinician as he practices the treatment of periodontal disturbances must have a clear conception of the development of the periodontal pocket and the purpose of treatment which is to remove the cause of the disturbance and eliminate the pocket.

GINGIVITIS

The objective symptoms of gingival inflammation appear where local irritants such as calculus, infection, open contacts, poorly adapted crowns, and inlays or fill-

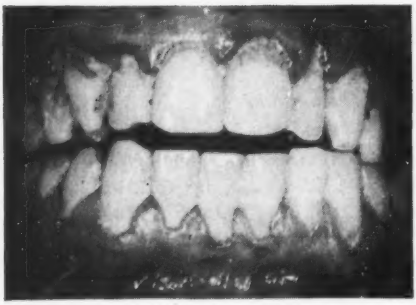


Fig. 1. Gingivitis. Lack of oral hygiene and home care.

ings encroach upon the free margin of the gingiva. In histological study of biopsies of the gingiva a much greater evidence of inflammation is observed than in the mouth of the patient and tissue free from inflammation is rarely found in microscopic study. Clinical appraisal of the damage sustained by the periodontium in any stage of advancing disease is usually far below what might actually be seen by the aid of the microscope.

Irritation of any kind lowers the resistance of the gingiva as it does in other tissues of the body. The ever present microorganisms of the oral cavity can be resisted more successfully by healthy tissue than by tissue in which the mechanism of defense is impeded by inflammatory exudates of a chronically irritated tissue. However one must not overlook the importance of the inflammatory reaction to injury and infection for this is primarily a defense reaction without which there would be no defense and no repair. Nature's effort to combat and throw off the irritant should always be aided by medicine and dentistry for, after all is said, it is nature that heals disease.

Fig. 2. Gingival hyperplasia. Girl of adolescent years. Edematous enlargement of gingiva with some fibrous changes in the tissue.

DIAGNOSIS AND TREATMENT OF GINGIVITIS

The diagnosis of simple gingivitis requires little discussion since it is characterized by redness and a tendency to bleed easily when traumatized. The cause is not easily determined since there may be several contributing factors such as the lack of oral hygiene, local irritants or local infection. The condition may be complicated by some intrinsic factors due to an unbalanced metabolism or impaired function of certain internal organs. To eliminate the cause of gingivitis one must first make a successful interpretation of the symptoms. Symptoms of gingivitis do not always respond to treatment readily and seldom respond to the treatment commonly called, "cleaning of the teeth." Meticulous scaling, medication to control infection, and stimulation of the blood supply, by intensive home care are necessary to effect a cure.

Fig. 3. Same as Fig. 2 after conservative treatment.

There are other forms of gingivitis which appear as oral manifestation of some sys-

temic disturbance among which are to be found edematous and hyperplastic gingiva.

HYPERPLASTIC GINGIVITIS

Gingival hyperplasia is quite common during the adolescent period of childhood. It is often quite resistant to treatment and requires considerable patience and persistence on the part of the dentist to bring about a satisfactory result. It is essential to recognize the difference between edematous swellings (inflammatory hyperplasia) that respond readily to treatment and fibrous hyperplasia of the gingiva[1] which does not respond without surgical treatment. It is not necessary to resort to surgery in edematous swellings but the fibrous

Fig. 5. Periodontitis simplex-Heavy calculus deposits of long standing.

type of hyperplastic tissue will seldom respond to local medication. Fortunately fibrous hyperplasia seldom occurs in the adolescent years, but if these edematous areas are neglected or overlooked during the early years fibrous changes gradually occur which increase the difficulty of successful treatment. The essentials of treatment include patience and persistence on the part of the operator and encouragement to the patient. Meticulous scaling, tissue stimulation, and medication must be regularly given to supplement good home care.[2]

PERIODONTITIS

Periodontitis is a progressive inflammatory condition which involves the superficial gingiva, the periodontal soft tissue, alveolar bone and the cementum. It may affect one or many teeth. The line of demarcation between chronic gingivitis and periodontitis is only a matter of degree as the deeper tissues including the bone marrow, alveolar bone, and periodontal tissue become involved. Weinmann[3] and others have shown the progress of inflammation passing along the vascular tracts into the bone marrow of the supporting bone in the intra-alveolar areas. Resorption of the alveolar bone gradually occurs shortening the alveolar crests which cause some periodontal membrane fibers to disappear and gradually the inflammatory infiltration in the surrounding tissue increases. Menkin[4] says that the accumulating exudate creates

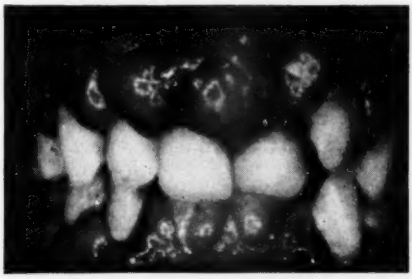


Fig. 4. Fibrous hyperplasia of the gingiva in man of 30 years. He said his gums had always been that way.

Fig. 6. Periodontitis complex. Young woman of 24 years in upper picture. Lower pictures show the areas at age 30.

what he has called a "lymphatic block" and this intensifies as the exudates increase in rapidly progressing periodontal disease. Inflammatory reaction increases bone destruction and connective tissue changes. These changes are followed by gradually deepening pockets. The tooth or teeth remain firm until, because of the loss of supporting tissue, they cannot sustain their position under occlusal stress. A tooth gradually becomes loose and movable as the time approaches when it causes annoyance and discomfort in mastication. Unless the progressive periodontitis is recognized and corrected before the bone loss is great enough to dangerously impair the functional capacity of the tooth the condition causes the loss of the tooth.

In periodontitis (simplex), which has progressed into deep pockets without being checked by treatment, frequent acute exacerbations occur when the surrounding tissue becomes infected by micro-organisms that proliferate in such pockets. Usually pus can be pressed out from such pockets where lateral or periodontal abscesses later occur. This advanced or exaggerated condition may still represent a purely inflammatory periodontitis of local origin, but responds more slowly to local treatment.

There is in all diseased processes of human body a certain combination of systemic predisposing factors and local factors which may cause an intensification of local disturbances. Boyle[5] has nicely expressed this as follows:

"Periodontal disease, like all other disease processes, is the end result of a combination of adverse local and systemic factors. Local factors probably in all instances are the immediate cause of the disease. The reactions of the tissues which tend to prevent invasion by bacteria or to protect against mechanical injury are always dependent for their effectiveness upon systemic factors." With the background of an impaired systemic functional balance a simple local disturbance may become complex.

PERIODONTITIS (COMPLEX)

In the comprehensive classification of Periodontal Disturbances by Orban[6] it is very clearly shown how progressive gingivitis extends into the periodontium and involves the periodontal fibers, alveolar and supporting bone. It is conceivable how a complex condition may develop from a case of simple periodontitis as the influence of gradually increasing systemic disturbances lowers the local tissue resistance thereby causing deep pocket formation and subsequent tissue changes.

The other form of complicated periodontitis given in the same classification is said to be secondary to periodontosis. By being secondary signifies that degenerative changes first occur in the local periodontal tissue. Degenerative changes cannot be observed clinically and such an entity is difficult to conceive in clinical terms. At times degenerative changes are believed to exist in the periodontal tissue when there is separation and drifting of one or more teeth from their normal position. The final phase in periodontosis is the sudden appearance of a deep pocket which opens the apparent healthy gingival sulcus into the deep affected periodontium. The opening of such a deep pocket immediately introduces oral fluids, infection, and the subsequent inflammatory reaction which classifies the condition as periodontitis complex. The occurrence of such deep pockets is usually characterized by the suddenness of their appearance and the lack of calculus in contrast to the usual presence of considerable calculus in the deep pockets of the progressive inflammatory conditions as they slowly develop into deep pockets. At this period of tissue changes there is little clinical difference between the two types except that one has developed slowly and the other rather suddenly. The latter entity has been described by Thoma[7] and Goldman as a syndrome resulting from a combination of both degenerative and inflammatory disturbances resulting from intrinsic and extrinsic factors.

Box[8] has recognized a necrotic form

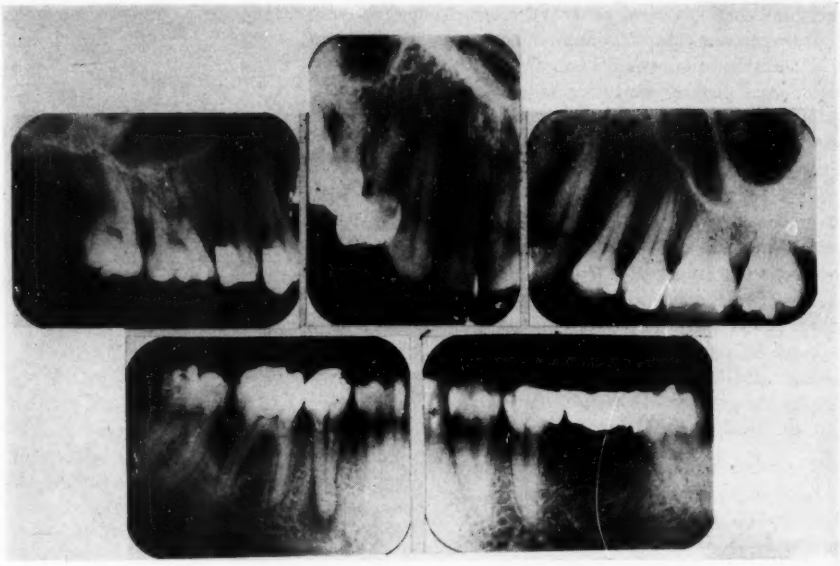


Fig. 7. Periodontitis complex, believed to be from degenerative changes in periodontosis following arrested tuberculosis.

of periodontitis which develops from tracts extending from the base of pockets or from gingival clefts into the deeper soft tissue. These tracts are interpreted as lymphatic vessels distended with a coagulum like material. The tracts carry toxic substances which possess a necrotizing action upon the soft tissue of the periodontium and destroy both cementoblasts and osteoblasts as well as to disorganize the connective tissue as the tracts spread apically. In this process of tissue necrosis the disorganized periodontal tissue produces symptoms from a local extrinsic factor that correspond to the degenerative changes which are usually associated with systemic factors without apparent local manifestation. This condition, however, is subject to systemic predisposing factors "that operate as assisting or preventing agents" in any tissue.

Ray and Orban[9] investigated 110 gingival biopsies and found necrotic foci in 75 of them. Foci were found where the patients were clinically healthy as well as in the periodontal tissue of those with a history of pregnancy, diabetes and various other diseases. They interpreted these findings of necrotic foci as degenerating and necrotic plasma cells, fibroblasts and disintegrating collagenous fibers. They found these necrotic foci in areas of chronic inflammation and concluded that they have some potentiality of leading to systemic disturbances because of the penetration of toxins into the deeper tissues and the possibility of their absorption into the blood stream. They did not observe the necrotic tracts of lymphatic channels described by Box. This is another important factor in the localization of the periodontal pocket.

DIAGNOSIS AND TREATMENT OF PERIODONTITIS

The diagnosis of periodontitis is made in several steps. The roentgenogram reveals a loss of more or less alveolar bone. The alveolar crests lose their pointed characteristic and abnormal translucent areas of

variable depth (vertical pockets) may appear around the roots of the teeth showing a loss of the supporting bone. When the periodontal pockets are tested by means of an explorer or probe the depth of the pockets should be recorded upon the examination chart. The translucent areas are studied to determine their significance in the plan of treatment. Since these translucent areas cannot always be reached by medication or surgery the prognosis must be considered before treatment is undertaken. However, one must not be too hasty to consider a condition hopeless. When the pockets are eliminated by treatment or surgery the progress of the disease is checked and the tissues should return to health.

The primary diagnosis in the complicated conditions that are characterized by the combination of both local and systemic factors is made from the following factors: the patient's history, the rapidity of the progress of the disease, the irregularity of the bone loss about the roots of teeth and the shifting position of teeth with deep periodontal pockets. These deep pockets often appear suddenly and are usually discovered by roentgenogram examination.

The cause of the systemic disturbance is much more difficult to determine and correct than the cause of the inflammatory conditions having only local factors to consider. The reflection of some degenerative or nutritional disturbance into the periodontal tissues is often suspected, but difficult to prove or to correct.

The effects of inflammation are usually easily observed by the character of the tissues and are easily corrected. When local irritants have been removed, infection controlled and the pockets eliminated by effective procedure, the factors that produce inflammatory disturbances are definitely removed. Meticulous home care in daily hygiene retains the key to a successful battle against further progress of periodontitis which represents a very large percentage of all cases of periodontal disease. When areas of lowered density lie in the bifurcation or trifurcation of molar teeth the prognosis is less favorable than when they lie in the interradicular supporting bone.

The treatment for any pathological periodontal pocket, regardless of its depth, is not complete until the pocket has been reduced to a minimum depth of approximately 2 millimeters.

Medication is beneficial in controlling infection and in stimulating better circulation in edematous tissues to revitalize them. Mouth washes of hydrogen peroxide and a metaphen solution of 1 in 2500 strength are useful in reducing acute infections. Superoxol and iodo-glycerol are recommended to help reduce soft, spongy gums to a pink and firm condition. Pocket elimination is essential to the successful conclusion of treatment. Gingivectomy or gum resection is the final resort to remove pockets and bring about a quick response in chronic inflammatory conditions surrounding pus pockets. The simplicity of the operation and the uniformity of the results following gingivectomy give it precedence in most cases.

Box*,[10] recommends the use of antiformin in pocket treatment. By local application it quickly reduces hemorrhagic gingiva from a state of chronic inflammation to normal color and firmness with closely adapted tissue to the cementum surfaces. He and his followers also have found a greater prospect of bone regeneration by this treatment where bony pockets have previously existed and much less need for surgical treatment.

Antiformin has a solvent action upon the epithelial lining of the pocket and it facilitates the thorough removal of all subgingival calculus which is often difficult to remove. It is needless to add that all septic material within the pocket is readily and thoroughly dissolved by the chemical action of the drug.

Antiformin is a strong alkaline solution which must be used with extreme care. It must not be allowed to touch any tissue

*Personal communication.

Fig. 8. Periodontal tissue showing Sharpey's fibers strongly attached to cementum in several layers of live cementum.

except the sulcus epithelium and root surface within the pocket. A drop at a time is carried into the pocket with a suitable instrument which can be carried to the bottom of the pocket. The antiformin is allowed to remain for a period of two minutes when it is neutralized with a 5 or 10% citric acid solution. After neutralization the pocket is thoroughly curetted and freed from calculus and debris. The area is now covered with a zinc oxide resin pack which is allowed to remain from three days to a week when the patient returns for examination and repeat of the treatment if necessary.[11]

In the classification to which reference was given[6] it will be noticed that about the same systemic factors are recorded in the etiology of inflammatory systemic gingivitis, periodontitis simplex, and complex as in periodontosis. The systemic background of both inflammatory and degenerative disturbances has been substantiated by various investigators referred to in the previous discussion. This bears out the fact that the systemic background usually influences the local manifestation of most periodontal disturbances and rarely ever can a purely systemic, clinically manifested periodontal pocket be found without some localizing factor. When local factors are corrected or obliterated and followed by intelligent and faithful home care by the patient it is well known that most periodontal disease is completely cured.

PERIODONTOSIS

The term periodontosis was recommended and accepted by the Academy of Periodontology in 1943.[12] It represents a degenerative, noninflammatory condition of the periodontal tissue which usually has no clinical manifestation at its beginning, but may be recognized occasionally by the unnatural drifting of a tooth from its normal position where there is no evidence of pocket formation or clinical evidence of inflammation in the periodontal tissue.

The concept of a disease of the periodontal tissue without clinical symptoms is not easily acquired. The wide range of clinical pictures of gross disturbances showing alveolar bone loss with deep periodontal pockets all labeled as periodontosis has led to considerable confusion as to just what is clinical periodontosis.

Periodontosis in its early stages is not a clinical entity, but a manifestation of intrensic factors affecting a local tissue. The most definite clinical evidence, which is sudden deep pocket formation, is the last stage in its development and at this point mouth fluids and infection enter the pocket and the usual inflammatory symptoms follow. It now becomes clinical periodontitis, but it is properly called periodontitis complex to distinguish it from a simple periodontitis which may or may not have intrinsic predisposing factors which is an inflammatory disturbance from its onset.

Periodontosis manifests itself in microscopic degenerative changes that take place in the connective tissue of the periodontal membrane resulting in a loss of alveolar bone followed by pathological drifting of teeth. It has only been observed in a few cases in histological study in its early stages. The early stages are said to pass quickly.[13] It may not be found clinically until a deep pocket becomes noticeable where only a few days or months previously no pocket and

no inflammation were evident. When the pathological pocket appears the condition assumes an inflammatory character superimposed upon the previously degenerated periodontal tissue and is now periodontitis complex.

ETIOLOGICAL FACTORS IN PERIODONTOSIS

It was believed by Gottlieb and concurred in by further investigation by Orban and Weinmann[13] that periodontosis may be due to the failure of the cementoblasts to form new deposits of cementum. This in turn is probably due to the degeneration of the periodontal connective tissue. Without new deposits of cementum the replacement of worn out periodontal fibers is not possible. The alveolar bone becomes resorbed from lack of stimulation and widened marrow spaces coalesce with the periodontal membrane resulting in a widened periodontal space.

It is assumed that there is a nutritional deficiency or metabolic disturbance that underlies the failure of the cementoblasts. Experimental evidence substantiates this assumption in that characteristic lesions have been produced in animals by starvation in Vitamin C. deficiency.[14, 15] However it is notable that the tissue breakdown is not initiated by the predisposing factor alone, but there is always a local factor of importance and the supporting tissues are unable to withstand functional stress.

Goldman[16] found lesions in the periodontal tissues of two spider monkeys that had died of amebic and bacillary dysentery. His description of the periodontal lesions resemble those described by Orban and Weinmann in their case of periodontosis. Although he found evidence of degeneration of the connective tissues in the periodontal membrane there was much soft calculus and bleeding with pockets present. The animals had had difficulty in eating. Perhaps these local factors should not be overlooked in their relation to the initiation of the disease or possibly the predisposing factors favored both periodontitis and periodontosis.

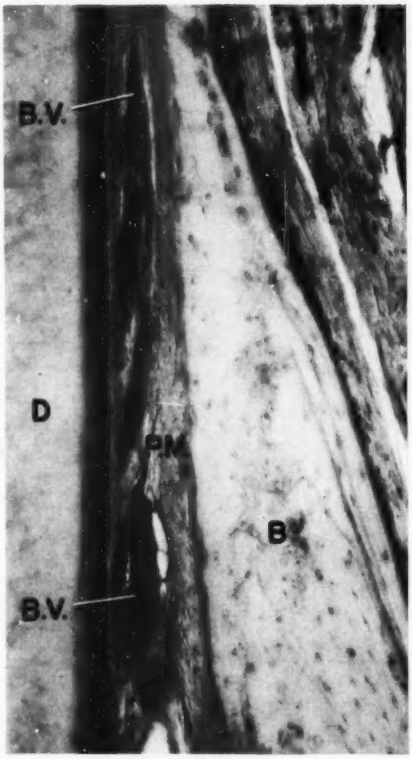


Fig. 9. Traumatism-thrombosis in blood vessels (BV) in periodontal membrane in the pressure area. B. bone, D. dentin.

Becks[17] found gingival inflammation, osteoporosis and infection in the supporting bone resembling periodontosis in dogs fed on a diet lacking in the filtrate fraction of Vitamin B complex and nicotinic acid. In his summary he makes the following statement:

"In areas where the inflammation has not reached the paradental bone structure a typical osteoporosis results with severe enlargement of all the largest capillaries, increased osteoclastic activity and replacement of bone by fat tissue." He also stated later that "endogenous factors can affect separately gingival tissue without involving primarily the osseous structure and vice versa."

In Gottlieb's case,[18] the first introduction of diffuse atrophy, the young patient suffered with an extreme case of influenza and pneumonia and probably some nutritional deficiency since it was reported soon after World War I.

In the case reported by Orban and Weinmann[13] the age of the patient and history are not given. The relation of the intrinsic predisposition to function is given as follows:

"As long as the systemic condition of the individual is perfect, the functional stress of the periodontal membrane is compensated by rapid new formation of fibers and cementum. However, if there are toxins circulating in the system or if the physiological metabolism is upset (endocrine dysfunction, nutritional deficiency, blood dyscrasias, toxins of micro-organisms, etc.) the reparative process is impaired and degeneration sets in. The presence of toxins in the system, or a disturbance of metabolism, may be primarily responsible for the disturbance of the balance in the dentition."

The relation of an endocrine imbalance is difficult to prove. However, dysfunction of the endocrine system has been observed in many patients suffering with periodontosis by Becks,[19] Goldman,[20] Boenheim,[21] and Weinmann.[22] The experiments of Schour[23] revealed changes in the periodontal tissues of hypophysectomized rats. It seems logical also to assume that the common occurrence of gingival hyperplasia in adolescent children who seem backward in development may represent some endocrine imbalance.

Diabetics usually have increased deposits of dental calculus, gingival inflammation with early vascular damage,[24] and are more susceptible to periodontal infections. All local factors greatly complicate the systemic predisposition manifested by rapid bone resorption about the roots of teeth. The frequent occurrence of periodontosis in diabetics does not justify the conclusion that this disease is the only intrinsic factor in that patient's history that may cause such loss of alveolar and supporting bone.[25] However, it is generally accepted that in any degenerative disease the periodontal tissues lose character and resistance and become more susceptible to injury and disease.

Talbot[26] attributed the occurrence of systemic periodontal disease to the absorption of toxic substances in gastro-intestinal disorders and observed that during periods of great physical stress oral manifestations of systemic disorders were most common when metabolic changes are most active. "These periods of stress are times when certain great life functions are developing or undergoing retrogression." These periods of stress include the teething period in infants, the adolescent period of youth, pregnancy of women and the climacteric period in advancing years of both men and women.

It seems to be evident from a study of reports in the literature that periodontal structures like other tissues under stress are affected by many systemic influences which alter their reaction to function and irritation. Periodontosis then, may we say, is brought about by disturbances or deficiencies of nutrition or some toxic influence commonly observed in gastro-intestinal disorders, degenerative and infectious diseases, endocrine imbalance, blood dyscrasia, avitaminosis or mineral starvation. With a predisposing weakness in the periodontium brought about by heredity or some nutritional deficiency or metabolic disturbance, tissue repair becomes impaired and degeneration may occur. As to the localization of the lesions it is very often that some obscure factor of local significance either functional or irritational is present.

DIAGNOSIS AND TREATMENT OF PERIODONTOSIS

The first clinical symptom of typical periodontosis is the shifting of a tooth or several teeth from their normal position. This drifting occurs without any sign of pocket formation or gingival inflammation. Central incisors separate or begin to rotate.

Fig. 10. Traumatism-pressure necrosis (N), in periodontal membrane, (PM), showing osteoclasts (OC) removing bone in backward resorption. (AB) Alveolar bone.

Frequently an incisor protrudes or elongates in abnormal eruption. At this time the disease is well on its way and other bone involvement may be found by roentgenogram. As a tooth drifts from its normal position it is often subjected to new and abnormal occlusal stress which gives the tooth an overload.

At a later period in the development of the disease deep pockets suddenly appear. These pockets may be buccal, lingual, or approximal intrabony pockets. They occur as the degenerated periodontal tissue extends to the gingival sulcus or when marginal inflammation is present and progresses rootward. Sometimes periodontitis and periodontosis occur simulaneously and appear as a symbiosis. In this condition the gingival pocket extends rootward until it reaches the area of degenerated periodontal tissue. The complete or final diagnosis cannot always be made at the first examination but as the treatment progresses a more complete understanding of the cases is possible. If the condition responds rapidly to local treatment it is evident that it is mostly inflammatory, but if it persists and bone involvement increases in spite of the removal of all local irritants, the intrinsic factors are still active that have not been controlled or corrected.

The treatment of periodontosis is more difficult than that given in the treatment of periodontitis. If treatment is begun before gingival inflammation and pockets are to be found there is little local treatment indicated other than to stabilize the drifting teeth with some mechanical splint and to adjust the occlusal relations to protect the affected teeth from severe lateral or protrusive stress. After careful study of the patient's history and general physical symptoms, cooperation by physician and dentist may bring some relief. A complete physical examination does not often reveal an abnormality of function in any organ, but there may be some clue to the systemic disorder. The general physical examination should include a thorough hematological examination with a determination of the blood level of Vitamin C and blood sugar. An analysis of the urine for sugar content or any evidence of abnormal kidney function, a blood count and hemoglobin index may reveal some intrinsic factor that needs attention. Blood dyscrasias cause changes in both the bony support and the soft tissue about the teeth. The basal metabolism test may reveal some endocrine disturbance which may be suspected from the patient's history and noticeable symptoms of a nervous disorder.

Boenheim,[27] who has cooperated in the medical care of many patients suffering with periodontosis, enumerates many therapeutic aids as follows:

1. Hormonal treatment when indicated to include the use of extracts and prepara-

tions of thyroid, thymus, pituitary, parathyroid, insulin, cortin and ovary.

2. Physical therapy, such as ice packs, light, radiation, electricity and diathermy.

3. Heavy metals including potassium arsenite, iron, copper and calcium usually prescribed in a combined form called heptogene.

4. Dietary control to eliminate the common autointoxication factors. Vitamins A, B, and C, gained mostly from large quantities of fruit consumed as well as milk products and liver.

In the treatment of the more common condition where deep pockets have formed and there is roentgenographical evidence of much bone loss about several or many teeth —a procedure much like that followed in treating periodontitis is indicated.

EXTRACTION OF HOPELESS TEETH

An appraisal of the prognosis must be considered and teeth that have lost so much supporting tissue that they cannot stand the demand of function and have drifted from their normal positions may require extraction. Pockets extending into the bifurcation or trifurcation of molar teeth become susceptible to periodontal or lateral abscess formation and are not, as a rule, responsive to local treatment or to gum resection, but palliative treatment is frequently indicated.

POCKET ELIMINATION

After meticulous scaling and intensive home care have reduced superficial inflammation (if present) and restored a more vigorous circulation in the gingiva, surgical treatment in the form of gum resection is recommended to eliminate all remaining pockets. The tissue overlying gingival and periodontal pockets is usually edematous or relaxed and lacking of a healthy tone. Scaling and medication with iodine, astringents and germicides will eliminate many pockets.

The objective of treatment is to reduce gingival pockets to a minimum and restore health and vigor to the tissue while the patient is trained in the proper home care of the tissue and teeth.

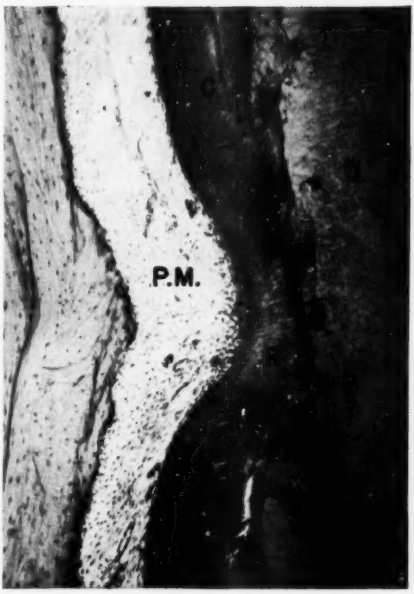


Fig. 11. Repair of resorbed area in root surface (R) and re-establishment of normal thickness of periodontal membrane(PM) by new layers of bone and cementum repair, (C). (D), Dentin.

The conservative method of subgingival scaling and curettage with constant attention to oral hygiene eliminates many pockets in a manner that leaves a minimum of root exposure following the treatment. Hirschfeld[28] says this method should be mastered by every practitioner and this writer heartily agrees with that statement.

TRAUMATISM

Periodontal tissues are very frequently traumatized during the process of mastication and more frequently during the mandibular excursions when no food is between the teeth. Gottlieb and Orban[29] demonstrated on dogs that pocket formation could not be caused by abnormal or excessive occlusal force. Traumatic injuries are

of an acute type and are followed by the usual inflammatory reactions, continued injury will result in a widening of the periodontal area and thickening of the periodontal tissue. If the traumatic force is discontinued the bruised and destroyed tissue will be resorbed and replaced with normal tissue in functional relation to the root and the alveolar process. This repair is of course dependent upon a normal metabolism to replace the tissues and upon the cellular mechanism to produce new deposits of cementum upon the injured surface of the root.

In patients suffering with periodontosis the teeth often drift into positions where there is an abnormal occlusal stress, especially during the lateral or protrusive movements of the mandible. Some teeth may erupt at a greater rate of speed than their neighbors and become subjected to excessive centric stress. With a weakened supporting structure and a degenerating periodontal membrane the tooth capacity for function is reduced proportionately.

The closing relation of the occlusal contact surfaces must be studied and often adjusted to permit free lateral and protrusive movements of the mandible. Lateral stresses are more often traumatic than the axial stress of centric occlusion. Irregularly worn occlusal surfaces and sharp edges should be corrected and careful spot grinding to relieve lateral pressure points should be a regular part of the periodontist's service.

Missing teeth should be replaced by artificial substitutes so that function may be restored in mastication and improper use of remaining teeth can be avoided. Compensation by new periodontal fibers and new deposits of cementum along with reinforcement of trabecular supporting tissue is a normal response to additional demands of occlusal stress. The response to occlusal stress upon teeth whose supporting mechanism is damaged by predisposing systemic factors or by any cellular weakness from any physical cause is not equal to functional requirements of usual stress so degeneration occurs.

In the treatment of periodontosis the removal of all local irritants, the effect of which upon the health of the supporting tissue is far greater than upon more normal tissue, is extremely important. Meticulous care must be given to prevent their presence. Improvement in home care and attention to dietary requirements, supplemented by concentrated vitamin administration and other medical treatment in cooperation with the endocrinologist will usually result in a favorable and encouraging response.

SUMMARY

The discussion of periodontal diseases was divided into those conditions in which chronic inflammation is the principal cause of the disturbance and those in which the predisposing factors lead to tissue degeneration and loss of function.

The inflammatory conditions discussed were chronic gingivitis including gingival hyperplasia and periodontitis.

Periodontitis complex was discussed as representing a syndrome or combination of the inflammatory condition and the degenerative condition acting at the same time or becoming superimposed one upon the other during the progress of the disease.

Periodontosis probably starts with the degeneration of some part of the periodontal tissue. The vitality of the cementoblasts seems to weaken and new deposits of cementum fail to be laid down. Periodontal fibers become separated and the affected tissue fibers lose their function and disappear. The loss of functional stimulation is followed by loss of alveolar bone.

It seems evident from a study of reports in the literature that periodontal structures are influenced generally by deficiencies in the nutritional supply and metabolism in the bone forming cells. Periodontosis may be the result of a disturbance or deficiency of nutrition or some toxic influence com-

monly observed in gastro-intestinal disorders, degenerative and infectious diseases, endocrine imbalance, blood dyscrasia, avitaminosis or mineral starvation. With a predisposing weakness in the periodontium brought about by heredity or some nutritional deficiency or metabolic disturbance, tissue repair becomes impaired and degeneration occurs.

The effect of local irritants upon the periodontal tissue is intensified when superimposed upon tissues with a lowered resistance.

The objective of treatment is to determine and correct the cause of the disease, to reduce gingival pockets to a minimum and restore health and vigor to the periodontal structures.

Conservative methods of treatment including meticulous scaling, control of infection, and intensive home care are to be recommended. Medication, nutritional instruction and occlusal adjustment should be given as indicated.

Pocket elimination by gum resection should follow when the desired result has not been attained because pockets will progressively deepen if not eliminated.

Systemic disturbances often may be the conditioning factor in local manifestation of tissue breakdown.

Sometimes a local disturbance in the gingiva can be corrected by treating the systemic factor as in Ascorbic Acid administration for gingivitis when there is evidence of a low blood level of Vitamin C.

No periodontal pocket can be cured by treating a systemic predisposing factor without local treatment for the pocket.

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REFERENCES

1. Hine, Maynard K. Fibrous Hyperplasia of the Gingiva. Jour. Am. Dental Association **44**:681, 1952.

2. Coolidge, Edgar D. Hypertrophic Gingivitis. Jour. Am. Dental Assn. **28**:1381, 1941.

3. Weinmann, Joseph P. Progress of Gingival Inflammation into the Supporting Structures of the Teeth. Jour. Periodont. **12**:71, 1941.

4. Menkin, Valey. Dynamics of Inflammation. The Macmillan Co., New York, 1940, p. 171.

5. Boyle, Paul E. Experimental Evidence of Systemic Influence on the Development of Periodontal Disease. Jour. Periodont. **22**:195, 1951.

6. Orban, Balint. Classification and Nomenclature of Periodontal Diseases. Jour. Periodont. **13**: 88, 1942.

7. Thoma, Kurt H., and Goldman, Henry M. Classification and Histopathology of Parodontal Disease. Jour. Am. Dent. Assn. **24**:1915, 1937.

8. Box, Harold K. Concerning the Pathogenesis of Periodontal Disease. Jour. Periodont. **19**:11, 1948.

9. Ray, Harold G., and Orban, Balint. Deep Necrotic Foci in the Gingiva. Jour. Periodont. **19**: 91, 1948.

10. Personal communication.

11. The technic of using antiformin is described in detail in five stages which should be thoroughly understood before attempting to use it. The detailed description may be found in the Journal of Ontario Dental Association of March, (vol. 30, No. 3, P. 84), 1953 by Keith F. Box. Also appears in D.D.S. **2**:407, 1953.

12. Report of the Committee on Nomenclature of the American Academy of Periodontology. Jour. Periodont. July supplement **14**:14, 1943.

13. Orban, Balint, and Weinmann, Joseph P. Diffuse Atrophy of the Alveolar Bone (Periodontosis). Jour. Periodont. **13**:31, 1942.

14. Boyle, Paul E., Bessey, Otto, and Wolback, S. B. Experimental Production of the Diffuse Alveolar Bone Atrophy Type of Periodontal Disease by Diets Deficient in Ascorbic Acid (Vitamin C). Jour. Am. Dent. Assn. & Dent. Cos. **24**:1768, 1937.

15. Glickman, I. Acute Vitamin C. Deficiency and Periodontal Disease. Jour. Dent. Res. **27**:201, 1948.

16. Goldman, Henry M. Periodontosis in the Spider Monkey. Jour. Periodont. **18**:34, 1947.

17. Becks, Hermann. The Effect of Deficiencies of the Filtrate Fraction of Vitamin B. Complex and of Nicotinic Acid in Teeth and Oral Structures. Jour. Periodont. **13**:18, 1942.

Discussion. Jour. Periodont. Supplement to Jan. 1944 issue, p. 48.

18. Gottlieb, B. Etiology and Therapy of Alveolar Pyorrhea. Ztsch. F. Stomatology **18**:59, 1920.

19. Becks, Hermann. Systemic Background of Paradentosis. Jour. Am. Dent. Assn. **28**:1447, 1941.

20. Goldman, Henry M. Periodontia. St. Louis, The C. V. Mosby Co., 1942, p. 185.

21. Boenheim, Felix. Pathogenic Importance of the Endocrine Glands in Paradontal Disease. Jour. Dent. Res. **17**:19, 1938.

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Psoriasis Gingivae*

by H. A. Brayshaw, Lt. Col., U.S.A.,** *Denver, Colorado, and* Balint Orban, M.D., D.D.S.,*** *Colorado Springs, Colorado*

PSORIASIS of the oral cavity has been reported on the lips, buccal mucosa, tongue and palate. Not all cases have been substantiated by histologic findings; some seem to have been confused with lichen planus or leukoplakia.

Max Scheer[1] states: "Psoriasis of the mucous membrane is so rare that few dermatologists claim to have recognized it as such, and probably many deny its existence. A routine examination I made of many psoriasis cases seen during a period of years, showed no lesions of the mucosa even remotely resembling psoriasis, except the case here reported. 'A man, age 26, salesman. Lips are dry, scaly, cracked and bleed readily. Lesions affected only the exposed portion of the lips. The scales were silvery, white and micaceous, and on gentle removal there was bleeding. A biopsy was not made from the lips.'"

McCarthy[2] states: "Solid, circumscribed, round or oval lesions the size of a dime have been designated as psoriasis nummularis. Split pea size, silvery appearance of scales is attributed to air content."

According to Usher,[3] "Psoriasis of the mucous membrane is one of the rarest of dermatological conditions. In 1903, Oppenheim reported the first cases with histologic examination. I examined the buccal mucosa of one hundred consecutive patients with psoriasis. Lesions of the buccal mucosa were noted in three patients. Histologic and clinical examination revealed that in one of these patients, a male—age 40, the condition was leukoplakia. The condition in the other two was psoriasis."

Oppenheim's[4] patient presented several plaques, entirely different in appearance from leukoplakia, on the buccal mucosa, the palate and the left palatal vault. He emphasized the importance of the presence of parakeratosis, acanthosis and papillomatosis.

MacKee and Foster[5] report a decrease in formation of melanin pigment in active lesions in psoriasis but no permanent disturbance in formation of melanin of pigment cells of the epidermis.

Burks and Montgomery[6] state: "We might interject here that failure to demonstrate fungi or bacteria in any case of psoriasis also fits in with opinion in general that psoriasis is not on a bacterial or fungous basis."

Goldman and Bloom[7] recently reported two cases: One, a 68-year-old man with a lip lesion; the second, a 28-year-old man with a lesion in the floor of the mouth. Both cases had psoriasis of the skin and scalp.

Three cases clinically observed and histologically identified are presented.

Case I—A 35-year-old colored man sought treatment for bleeding gums. Oval lesions of about 1 cm. in diameter slightly elevated were found on the papillary and attached gingiva bilaterally, just distal to the upper cuspids (Fig. 1). Minute multiple bleeding points resulted from light stroking with the smooth, curved side of an explorer. The scaly surface presented a glistening nummular appearance with a slight diminution in pigmentation. Both lesions were excised completely and the areas healed uneventfully without recurrence after nine months. This patient presented no skin lesions.

Case II—A 33-year-old man of Irish de-

*Paper presented at the 30th Annual Meeting of the International Association of Dental Research, March 23, 1952—Colorado Springs, Colorado.

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Fig. 1. Case I. Patient colored, male, 35 years of age. Oval lesions between maxillary cuspid and first bicuspid. Pin-point bleedings upon slight scraping. Small, silvery scales on surface.

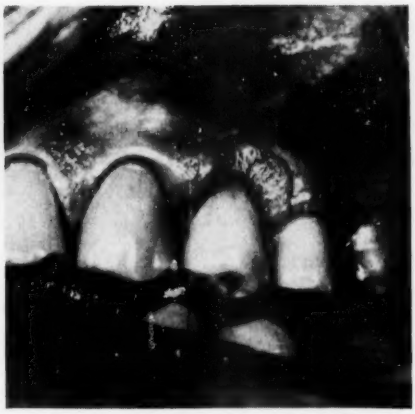

Fig. 2. Case II. Patient white, male, 33 years of age. Crescent-shaped lesion above first maxillary bicuspid. Whitish, irregular depression.

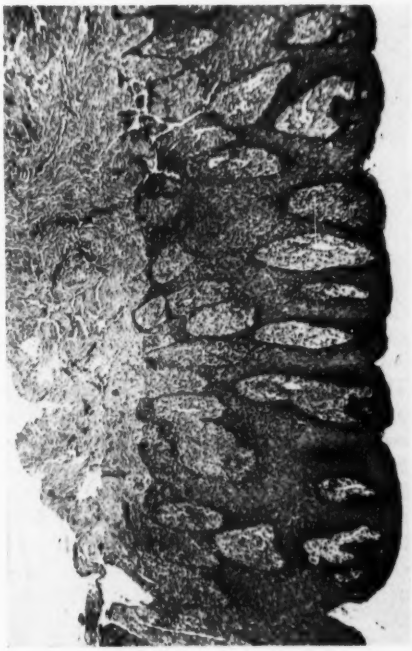

Fig. 3. Biopsy specimen of Case I. Elongated epithelial ridges; lacy fusion between epithelial pegs; clubbing of connective tissue papillae; thinning of suprapapillary epithelium.

Fig. 4. Higher magnification of Figure 3. Thin epithelium; parakeratosis; small microabscesses with migrating leukocytes.

Fig. 5. Biopsy specimen of Case II. Elongation and clubbing of epithelial ridges. Thick layer of parakeratotic cells on irregular surface.

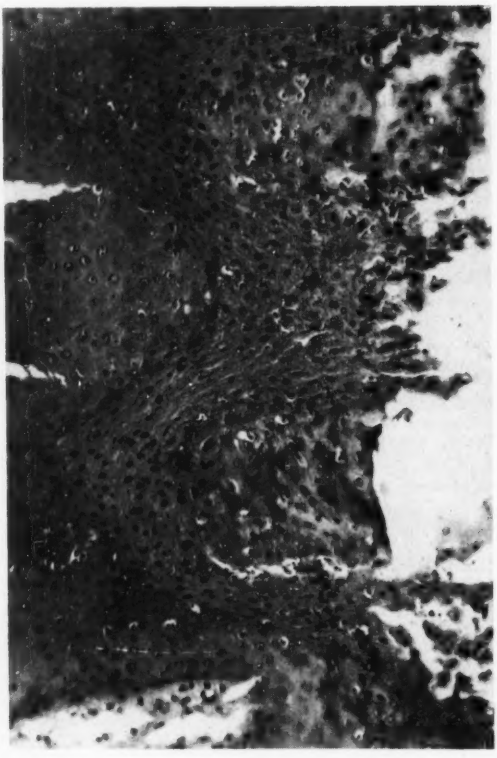

Fig. 6. Higher magnification of Figure 5. Parakeratotic cells forming surface; shedding of cells; small microabscesses; loose connection between parakeratotic cells.

scent complained of a whitish-gray crescent-shaped lesion on the attached gingiva over the upper left first bicuspid (Fig. 2). It was of indefinite duration and unknown origin. There were no psoriatic lesions on the body surface. The oral lesion bled easily on contact. It was entirely excised for biopsy study and healed uneventfully.

The biopsy specimen of the first case reveals an increase in the length of the epithelial ridges (Fig. 3). The epithelial ridges become fused and appear in a lace-like pattern. The connective tissue papillae are infiltrated by inflammatory cells and widen in a club-like manner toward the surface. The capillaries in the connective tissue papillae appear dilated. The supra-papillary epithelium is thin, consisting of only a few layers of epithelium (Fig. 4). The intra-epithelial spaces seem to be widened and numerous polymorphonuclear leukocytes are migrating toward the surface. The surface of the epithelium is parakeratotic and small microabscesses are seen along the surface. The inflammatory cells in the deeper papillary layer are plasma cells and lymphocytes. The reticular layer of the lamina propria is free of inflammation.

The biopsy specimen of the second case shows a circumscribed area of a parakeratotic lesion with elongated, club-shaped

Fig. 7. Case III. Biopsy shows gingival margin. Hyperkeratotic layer with microabcesses beginning at margin and extending into attached part of gingiva.

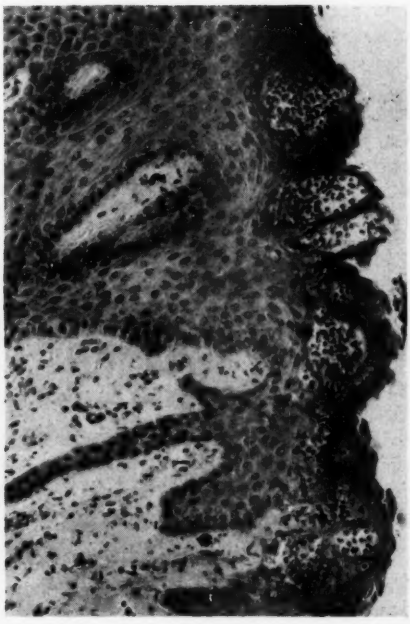

Fig. 8. Higher magnification of surface, showing heavy parakeratotic layer and rather large microabscesses filled with leukocytes.

epithelial ridges and pegs (Fig. 5). The epithelium is thickened, except in the suprapapillary region, where the epithelium is thinned to a few epithelial cell layers which separate the connective tissue from the parakeratotic layer. The parakeratotic layer is extremely thick. The cells show the typical pyknotic appearance. These cells are loosened in their connection and harbor small microabscesses. The surface shows considerable desquamation (Fig. 6).

Case III—The third case is submitted without a clinical photograph, but its appearance was similar to the previous cases. The gingival margin between the two upper bicuspids showed an opaque, whitish area of about 2 mm. in diameter. The surface was irregular and showed small, silvery, scale-like spots. The biopsy specimen (Fig. 7) shows an area of parakeratosis starting at the gingival margin, extending toward the attached gingiva. The heavy layer of parakeratotic cells is studded with variably-sized microabscesses filled with leukocytes (Fig. 8). In the center of the area, the connective tissue papillae reach close to the surface. Some papillae are covered only by a parakeratotic layer without normal epithelial cells. There is only a very slight inflammatory reaction in the subepithelial connective tissue.

DISCUSSION

Psoriasis is a skin disease of chronic inflammatory nature and unknown etiology. Lesions of the oral mucosa have been described in the literature, but lesions of the gingiva have not previously been reported. This paper presents three cases, studied histopathologically as well as clinically.

Clinically, the lesions of all three cases appeared as small plaques. The lesion in the first case was bilaterally symmetrical. The surface in all these cases was irregular, covered by small, silvery scales. Tiny bleeding points appeared upon slight scraping of the surface with a dull instrument. There were no subjective symptoms, other than a vague, itchy feeling.

Histopathologically, psoriasis is characterized by: 1. Parakeratosis; 2. Elongation —clubbing and lacy fusion of the epithelial ridges and pegs; 3. Thinning of the suprapapillary part of the epithelium so that often only a parakeratotic layer covers the papilla; 4. Widening of the tip of the connective tissue papillae due to inflammation, widening of the capillaries and edema; 5. Presence of microabscesses located in the parakeratotic layer of the epithelium.

These features were present in varying degrees. In all three cases, the numerous fine points of bleeding are explained by the parakeratotic surface lying directly above the hyperemic connective tissue papillae. The parakeratotic layer is easily scraped off, exposing the connective tissue.

SUMMARY AND CONCLUSIONS

Three cases of psoriasis of the gingiva are reported. The clinical features are explained by the microscopic picture. Parakeratosis, proliferation, clubbing and lacy fusion of the epithelial ridges, hyperemia and edema in the connective tissue papillae, thinning of the suprapapillary epithelium and microabscesses are the characteristic features.

REFERENCES

1. Scheer, Max, "Psoriasis of the Mucous Membrane of the Lips"—Archives of Dermatology and Syphilology 9:594, 1924.

2. McCarthy, Lee, Histopathology of Skin Diseases—Pages 194-199 Mosby, 1931.

3. Usher, B., "Psoriasis of the Mucous Membranes." Arch. Derm. and Syph. 28:488, 1933.

4. Oppenheim, M., "Psoriasis Mucosae Oris," Monschr. für Pract. Dermat. 37:48, 1903.

5. MacKee, G. M., and Foster, P. D., "Histopathogenesis of Psoriasis and Its Aberrant Lesions." Arch. Derm. and Syph. 34:35, 1936.

6. Burks, J. W., and Montgomery, H., "Histopathologic Study of Psoriasis." Arch. Derm. and Syph. 48:479, 1943.

7. Goldman, H., and Bloom, J., "Oral Manifestation of Psoriasis"—Oral Surg., Oral Med., and Oral Path. 4:48, 1951.

. . . . . *DENTIFRICE MAKER, clean thy house.* Present day advertisements for dentifrices are noted chiefly for their flagrant disregard for the truth and their low ethical standards. In recent years, the hucksters in the dentifrice field have become bolder with each new magazine ad and radio and television commercial until the false and misleading claims for dentifrices have become a public menace.

The American people are being led to believe that dentifrices will prevent dental disease, overcome mouth odors and help to cure bleeding and unhealthy gums. Evidence has not been published in any recognized scientific journal to justify these claims.

These claims are false or, at best, misleading. I do not know of a dentist—private practitioner or research scientist—who believes these claims to be true. There are no indications that the dentifrice industry is willing to correct this unwholesome and dishonest situation. The time has come for the dental profession to take action. The solution is simple. Dentists should recommend to their patients that they select a dentifrice on the basis of its cleansing properties only and not invest their money in a product for which the manufacturer's claims cannot be supported. One criterion for determining the honesty and authenticity of manufacturing claims is the product's eligibility for advertising or exhibit in media of the American Dental Association.—*From May 8 "News Letter," by Allen O. Gruebbel, D.D.S., former Secretary, Council on Dental Health, American Dental Association.*

. . . . . This preliminary study indicates that there is no standardization of toothbrush textures but there should be. At present if a dentist recommends a certain type of brush, there is little assurance that his patient will get the texture desired.—*From "Variation in Tooth Brush Textures," by M. K. Hine, Jr. J.A.D.A. 46:536, 1953.*

The Effect of the Systemic Administration of Cortisone Upon the Periodontium of White Mice

by IRVING GLICKMAN, IRVING C. STONE, AND TRIBHAWAN N. CHAWLA, *Ziskin Memorial Research Laboratory, Department of Oral Pathology and Periodontology, Tufts College Dental School, Boston, Mass.*

CORTISONE (11-dehydro-17 hydroxycorticosterone), Kendall's Compound E, is one of many closely related and qualitatively different, steroid derivatives produced by the adrenal cortex.[1] Among the effects attributed to the systemic administration of cortisone in experimental animals are the following: stimulation of protein catabolism, increased carbohydrate blood levels at the expense of tissue proteins, increased nitrogen and potassium excretion,[2] retardation in the healing of wounds,[3-4] and retardation in growth.[5]

With excessive administration of adrenocorticotropic hormone (ACTH), a substance which stimulates adrenal secretion and has many effects in common with cortisone, oesteoporosis has been reported in humans;[6] and osseous changes, consisting of reduction in chondrogenesis and osteogenesis in the proximal epiphyseal cartilage, reduction in the number of large vacuolated cartilage cells, reduction in the number of osteoblasts, irregular bony trabeculae and wider marrow sinusoids, were described in experimental animals.[7]

Although the effects of the systemic administration of cortisone upon numerous body tissues have been described, changes in the periodontal tissues have not been considered. A study was therefore undertaken to investigate the effect of systemic administration of cortisone upon the periodontal tissues of experimental animals.

EXPERIMENTAL PROCEDURE

Twenty male and female littermate white mice 4-6 weeks old, weighing 16-22 grams were used as experimental animals. Ten animals were injected intramuscularly every twenty-four hours with 0.5 mg. cortisone (Cortone, Merck). This dosage was selected after preliminary experimentation which indicated that mice could not be maintained beyond a two to three week period when daily dosages of one or two milligrams were injected. Animals injected with saline and uninjected animals served as controls.

The animals were maintained for a forty-three day experimental period. Eight survived and two died between the twenty-seventh and thirtieth days. The animals were sacrificed at the end of the experimental period. At autopsy, the adrenals were slightly smaller than the controls and no gross liver changes were observed. The maxilla, mandible, vertebrae, and femur were removed and prepared for histologic study.

GROSS OBSERVATIONS

The cortisone-injected animals appeared less active than the controls. At time of sacrifice these animals were smaller than the controls, showed some hair loss on the snout, while the hair covering the remainder of the body was thinner. There were no grossly detectable changes in the oral tissues of the control or cortisone-injected animals.

The *gingivae* of both the control and cortisone-injected animals presented a comparable number of isolated areas of marginal inflammation associated with local irritation from food particles. The destruction of the interdental gingiva associated with the inflammation appeared to be more severe in cortisone-injected animals. In those interdental gingival papillae devoid of appreciable inflammatory involvement, the collagen fibers were thinner and less densely arranged in the cortisone-injected animals.

MICROSCOPIC OBSERVATIONS

CONTROL ANIMALS

Alveolar Bone-Interdental Septa

The interdental bone between the molars was lined on one aspect by a thin layer of osteoid and adjacent osteoblasts (Figures 1 and 3). Along the other aspect, the bony border was smooth in some areas and irregularly indented in others. An occasional multinuclear osteoclast was seen in concavities along the surface. At the gingival crest of the bone, a small section of osteoid was seen.

The *periodontal membrane* was densely collagenous with well-formed fibroblasts.

THE BIFURCATION AREA

In the bifurcation area of the molars, a broad peripheral zone of newly formed bone was seen adjacent to the periodontal membrane, demarcated from the underlying bone by a deeply staining appositional line (Figure 5). Osteoblasts were aligned along the periphery, separated from each other by collagenous fibers embedded in the bone.

The *periodontal membrane* consisted of densely arranged fibers with numerous, well-formed cells.

CEMENTUM

The cementum was thin and acellular in the gingival half of the roots and bulbous and cellular in the apical region. In many areas, a thin peripheral border of cementoid was seen along the apical portion of the root.

CORTISONE-INJECTED ANIMALS

Alveolar Bone-Interdental Septa

The interdental septa were reduced in height; the margins of the septa stained deeply and presented a minutely granular border (Figures 2 and 4). There was no appreciable evidence of osteoblasts or osteoblastic activity. In a few isolated areas, shrunken, deeply staining osteoblasts were seen partially surrounded by faintly staining osteoid. The bone cells appeared enlarged compared with normal. The appositional lines were less clearly marked and in some instances were unusually irregular.

The *periodontal membrane* was edematous. The fibroblasts were markedly reduced in number and shrunken in appearance. The collagen fibers were reduced in amount, wavy, and fibrin-like and separated by the edema.

THE BIFURCATION AREA

The bone was outlined by a deeply-staining irregularly indented border (Figure 6). With the exception of a small area of new bone formation and a few poorly staining osteoblasts at the crest, there was no evidence of peripheral bone apposition.

The *periodontal membrane* was edematous. The fibroblasts were markedly reduced in number and shrunken in appearance. The collagen fibers were reduced in amount, wavy and fibrin-like and separated by edema.

CEMENTUM

In some areas the cementum appeared thinned and devoid of newly formed cementoid. However, the finding was not consistent or notable enough to be definitive.

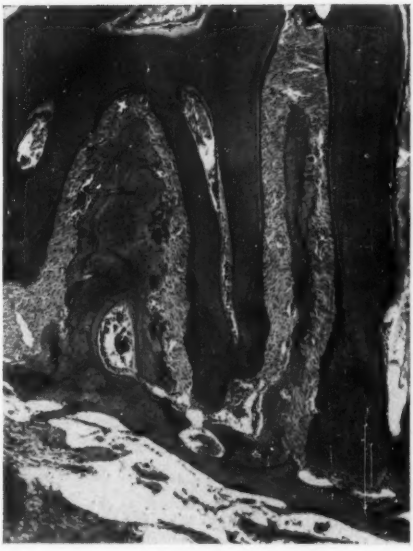

Fig. 1. Control animal. Mesio-distal survey section through the mandibular molar area showing molar roots and alveolar bone. (H. & E. × 88)

Fig. 2. Cortisone-injected animal. Mesio-distal survey section through the mandibular molar area showing molar roots and alveolar bone. (H. & E. × 88)

Fig. 3. Control animal. Detailed study of interdental septum and periodontal membrane shown in Figure 1. There is a thin layer of osteoid bordered by a row of osteoblasts along one surface of the septum (left). The other surface (right) presents concavities of resorption with an occasional osteoclast. (H. & E. × 350)

Fig. 4. Cortisone-injected animal. Detailed study of interdental septum and periodontal membrane shown in Figure 2. Note irregularly indented, deeply staining bone margin and absence of osteoblasts and osteoid. The fibroblasts of the periodontal membrane are reduced in number and the collagen fibers appear fibrin-like and fragmented (Compare with Figure 3). (H. & E. × 350)

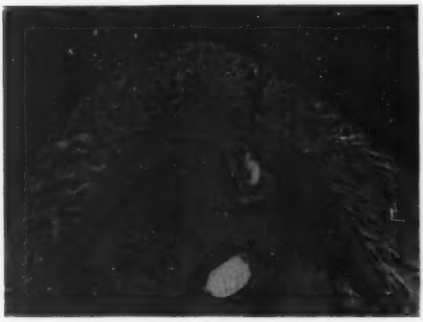

Fig. 5. Control animal. Detailed study of bifurcation area of the molar shown in Figure 1. Note the broad peripheral zone of recently formed bone which is demarcated from the underlying bone by a deeply staining appositional line. (H. & E. × 350)

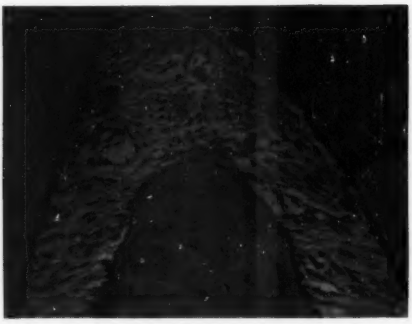

Fig. 6. Cortisone-injected animal. Detailed study of bifurcation area of the molar shown in Figure 2. Aside from a small area of osteoid at the crest, there is no apposition of newly formed bone. Instead, the margin of the bone is formed by a thin, deeply staining, irregularly indented line. Note the edematous appearance of the periodontal membrane, the diminished number of cells, and the fibrin-like appearance of the collagen fibers. (H. & E. × 350)

In the *vertebrae* and *femur* of the cortisone injected animals, the bony trabeculae were thinned and presented irregularly indented margins. Bone formation, normally seen in relation to the periosteum was markedly diminished or absent (Figures 7 and 8). The periosteum was thin and the collagen fibers reduced in number, wavy and fibrin-like. The number of fibroblasts was markedly reduced and the normal morpho-differentiation to form osteoblasts in relation to the adjacent bony surface was not seen.

DISCUSSION

From the periodontal viewpoint, the osteoporosis of alveolar bone, absence of appreciable new bone formation and the accompanying reduction in the height of tooth-supporting bone are particularly noteworthy. On a theoretical basis, explanation for the osteoporosis may lie in either (a) a cortisone induced accentuation of protein catabolism in the bone matrix[2] or (b) a cortisone induced inhibition of the normal anabolic processes (Albright) or (c) a combination of both phenomena. The extent to which excessive catabolism of the protein of the bone matrix might account for the osteoporosis could not be evaluated by experimental methods employed in this study. However, the reduction in the number of osteoblasts and amount of newly formed bone matrix are in conformity with Albright's interpretation of osteoporosis based upon impairment[8] in physiologic bone anabolic activity.

According to Albright,[9] among the steroid hormones produced by the adrenal cortex, are two types, the "Nitrogen" or "N" hormones which stimulate protoplasmic anabolism, and the "Sugar" or "S" hormone which inhibits this activity. Normally, in the male and female, these hormones are in balance. Repeated injections of cortisone such as were administered in this experiment could create an excess in "S" hormones with impairment in deposition of osteoid by the osteoblasts, and a resultant osteoporosis. The osteoporosis of Cushings Syndrome and of the General Adaptation Syndrome of Selye as well as that following systemic administration of ACTH are all explained by Albright on the basis of increase in adrenal cortical "S" hormone.

In the periodontal membrane of the cortisone injected animals, the alterations in the fibroblasts and collagen fibers are comparable to those described in connective tissue in other areas of the body in

Fig. 7. Control animal. Lateral surface of the femur in the region of the metaphysis showing the periosteum, underlying bone and bone marrow. Note the morphodifferentiation of the periosteal cells to form osteoblasts along the bone margin, and the formation of osteoid in areas of indentation indicative of previous bone resorption. (H. & E. × 600)

Fig. 8. Cortisone-injected animal. Lateral surface of the femur in the region of the metaphysis showing the periosteum, underlying bone and bone marrow. The collagen fibers of the periosteum are reduced in number, wavy and fibrin-like in appearance. The number of connective tissue cells is markedly reduced. The underlying bone is thin as compared with the control (Figure 7). The bone margin is irregularly indented and devoid of osteoblasts or osteoid. (H. & E. ×600)

experimental animals subjected to systemic administration of cortisone.[10] The cortisone apparently inhibits the fibroplasia which is necessary for the normal maintenance of the periodontal membrane and thereby produces marked structural changes.

It should be noted that bone and periosteum in other areas of the skeletal system presented changes similar to those in the alveolar bone and periodontal membrane. This interrelationship is consistent with the often repeated observation in experimental animals that the alveolar bone and periodontal membrane reflect the condition of the remainder of the skeletal system in systemically induced osseous disturbances.[11]

The comparatively greater severity of destruction of the inflamed interdental gingival papillae in the cortisone-injected animals is an interesting finding. However, interpretation of its significance is subject to the qualifying limitation imposed by the uncontrolled variation in the intensity of the local irritating factors. The changes in the gingival connective tissue in areas uninvolved by inflammation were comparable but less severe than the changes in the periodontal membrane of the cortisone-injected animals.

In general, despite the striking nature of the changes in the cortisone-injected animals, no inference regarding the effects of therapeutic systemic dosages of cortisone upon the periodontium of humans may be drawn from our findings. Determination of the latter information would require experimentation with a broad gradation of appropriately calculated dosages which is beyond the scope of the present study.

CONCLUSIONS

In mice subjected to daily injections

with 0.5 mg. of cortisone for periods up to 43 days, microscopic examination of the periodontal tissues revealed osteoporosis of alveolar bone characterized by reduction in the number of osteoblasts and amount of newly formed osteoid matrix; reduction in the height of alveolar bone, edema of the periodontal membrane with reduction in the number of fibroblasts and collagen fibers, and degeneration of the collagen fibers. These changes occurred unrelated to gingival inflammation which occurred in isolated areas associated with local irritation. The changes in the alveolar bone and periodontal membrane were comparable to those in the bone and periosteum in other areas of the skeletal system.

REFERENCES

1. Kendall, E. C., Mason, H. L., Myers, C. S., and Allers, W. D.: "A Physiologic and Chemical Investigation of the Suprarenal Cortex." J. Biol. Chem. **114**:56, 1936.

2. Long, C. N. H., Katzin, B., and Fry, E. G.: "The Adrenal Cortex and Carbohydrate Metabolism." Endocrinology **26**:309, 1941.

3. Ragan, C., Howes, E. L., Plotz, C. M., et al.: "The Effect of ACTH and Cortisone on Connective Tissue." Bull. New York Acad. Med. **26**:251, 1950.

4. Lattes, R. J., Bunt, J. W., Rose, H. M., Jessar, R. A., Villancourt, D. G., and Ragan, C.: "Lack of Cortisone Effect in the Early Stages of Inflammation and Repair." Am. J. Path. **29**:1, 1953.

5. Wells, B. B., and Kendall, T. C.: "The Influence of Corticosterone and C 17-hydroxydehydrocorticosterone (compound E) on Somatic Growth." Proc. Staff Meet. Mayo Clinic **15**:324, 1940.

6. Sprague, R. G., Power, M. H., Mason, H. L., Albert, A., Mathieson, D. R., Hench, P. S., Kendall, E. C., Slocumb, C. H., and Polley, H. F.: "ACTH on Man." Arch. Int. Med. **85**:199, 1950.

7. Becks, H., Simpson, M. D., Li, C. H., and Evans, H.: "The Effect of Adrenocorticotrophic Hormone (ACTH) on the Osseous System in Normal Rats." Endocrinology **34**:305, 1944.

8. Albright, F.: "Osteoporosis." Ann. Int. Med. **27**:861, 1947.

9. Albright, F., and Reifenstein, E. C.: "The Parathyroid Glands and Metabolic Bone Disease." Williams and Wilkins, 1948, p. 183.

10. Zoger, Seymour: "Observations on the Influence of Cortisone on Tissue Response in Injury." Yale Jour. Biol. & Med. **25**:202, 1952.

11. Glickman, I.: "The Experimental Basis for the 'Bone Factor' Concept in Periodontal Disease." J. Periodont. **20**:7, 1949.

DR. CELIA RICH

1878 — 1953

Dr. Celia Rich, well-known for her activities in the field of Periodontology, died on April 12, 1953 in Vanderbilt Hospital, Nashville, Tenn. Dr. Rich became ill after returning last fall from a tour abroad and although she had apparently improved, her condition became progressively worse during the month of April.

Dr. Rich's many contributions to dentistry are well known. She was graduated from Vanderbilt University Dental School and received many honors and responsibilities in organized dentistry. She was the first President of the National Association of Women Dentists in 1922-23, and was President of the Nashville Dental Society in 1944.

For many years she was an associate editor of the Journal of Periodontology and had been honored by election as a Fellow in the American Academy of Periodontology. Dr. Rich was very active in dental organizations and made many contributions to the progress of dentistry in general and periodontology in particular. Her quiet, sincere and pleasant personality made her very popular, and she leaves many friends who were inspired by her.

Pre-Operative Procedures and Post-Operative Expedients in Periodontal Surgery

by RAOUL H. BLANQUIE, D.D.S., *San Francisco*

THE degree of satisfactory results, following periodontal surgery, or any other procedure involving the principle of pocket eradication, bears an intimate relation with the preliminary care and attention given to the tissues that are to be eliminated.

The principle of these various methods which, besides surgery, includes chemical and electro therapies, is to remove or destroy the soft diseased tissues of the lesion or pocket and replace them, through the process of regeneration, with healthy investing tissues having shallow gingival invaginations adhering closely and uniformly over the crests of the alveolar bone. These procedures will attain their objectives under more favorable conditions when they are preceded by adequate measures to eliminate, or at least reduce perceptibly, the inflammatory and infective changes within the periodontium.

The approach to these methods of periodontal treatment too often displays the attitude that they may be carried out without consideration being given to the local symptoms of periodontal disease as they relate to the condition of the field of operation, the post-operative comfort of the patient, the normal recovery of the new tissues and the possibility of dissemination of the infection. This failure has often been the cause of unsatisfactory results for which the particular modus operandi was held responsible. It is generally contra-indicated to cut into or remove tissues in which inflammation and infection are present. These symptoms have a particular significance in pocket eradication therapies as unabated they may be responsible for profuse bleeding, undue post-operative pain, excessive granulation tissue with the possibility of new pocket formation, prolonged healing period and a bacteremia with chances of secondary involvement. The additional time that precautionary measures may occasion will more than be compensated by the preclusion of unfavorable factors that otherwise might obtain.

The removal of loose detached tissue, wherein inflammation is present, cannot be readily accomplished with any degree of accuracy. In the surgical method the difficulty is increased by the accelerated bleeding which will impede the vision of the operating field. The application of the surgical dressing also is hampered by the incorporation of blood in the material which tends to weaken its composition and make it more liable to become loose or fall off. The ensuing failure to pack properly, especially in the interproximal spaces, will generally cause the increase of any post-operative pain and discomfort. Loose packing will promote irritation to the regenerating tissue through its sharp moving edges and by permitting food to lodge under it.

In both periodontitis simplex and complex, which constitute the majority of periodontal cases, the inflammatory processes in the soft tissue walls of the pocket will be found to extend beyond the limits of tissue removal and into contiguous tissues that have normal attachment. By leaving inflamed tissue in situ the new granulating tissue will be susceptible to these inflammatory processes to the extent of reproducing similar conditions in it. This new tissue, if left to its own devices, may terminate in the reestablishment of the former pockets or, at least, with edematous gingival margins.

The practice of removing periodontal pockets, without first arresting the infective changes present, subjects the patient to a possible bacteremia.[1, 2, 3, 4] Several investigators have reported the possibility of such a result simply from the massage or the manipulation of infected gums.

While they believe that such a bacteremia is transient and perhaps harmless in a normal individual, they are also of the opinion that in an individual with valvular heart disease, such bacteremia may localize on the heart valves and produce subacute bacterial endocarditis. In the absence of evidence to the contrary, it might be inferred that a bacteremia, following periodontal surgery or any periodontal manipulation, may produce disorders or aggravate existing ones in individuals not in good health or, at least, with a resistance below normal. It is a known fact that operating upon a periodontal pocket, where the infection has not been arrested, and in a patient suffering from a systemic ailment of an infective nature, an exacerbation of symptoms of that particular condition will often follow. Such a reaction would seem to indicate that a relation exists between the two disorders.

Mitchell[5] and Helman have stated that "as bacteremia does result from oral manipulations in the presence of oral sepsis and since bacteremia has been known to be detrimental to some patients and potentially dangerous to others . . . it would be folly to tolerate oral sepsis. This conclusion does not imply that an oral focus should be regarded as 'anything readily accessible to surgery' . . . and that in effort to prevent bacteremia precautionary measures should be taken prior to manipulations."

The periodontal pocket, as a source of focal infection, has received but relatively recent attention in contrast with that given to peri-apical infections in the past. This lesion is a site of infection open to both blood and lymphatic channels in the presence of high bacterial activity. Periodontal[6] infections are now claimed to be the most serious of all oral infections by reason of their heavier flora, their proximity to blood and lymph streams and the extent of area involved.

Whatever doubt, regarding the focal infection theory, that may have been caused by the report of the University of Michigan Group,[7] under the general direction of the Council on Dental Health of the American Dental Association, should have been dispelled by the resolution[8] adopted by the American Society of Oral Surgeons at their meeting in Washington, D. C. in October 1951, with 335 members present, unanimously condemned not only the deductions but the right of the Council to accept as authoritative the opinion of one group of individuals in one center, and stated that a policy should never be formulated from unsound deductions or inferences. The viewpoint on this subject of probably the majority of practicing physicians and dentists, was well expressed by John A. Kolmer,[9] professor of medicine at Temple University, who said that "Focal infection as a potential cause of systemic conditions and diseases is no longer a theory but a principle or concept firmly established in both medicine and dentistry." The essayist would add his belief to this opinion and based upon thirty years of teaching and clinical experience with periodontal disorders. Aside from the clearing up of more serious symptoms, most patients, following treatment, have expressed the feeling of renewed vitality and the loss of a tired sensation which could have been caused by the continual absorption, over the period of many years, of bacterial toxins from the periodontal lesions.

The elimination, or adequate reduction, of inflammatory and infective processes within the periodontium and establishing hygienic conditions therein, are best accomplished through medication, sub-gingival curettage and the relief of excessive stresses upon the teeth. These three measures may be considered pre-requisites in practically all cases of periodontal treatment in which pocket eradication is indicated.

An invaluable aid in bringing about the desired results is the use of the Vastine[10] method of medication whereby suitable germicidal agents are pumped, by means of a rubber cup mounted on a handle, under the free margin of the gingivae throughout the lesion and within the soft tissues. Vastine has recommended the use of aniline

dyes which are effective in the reduction of both inflammation and infection. An understanding of this method is advisable as its improper application could result in forcing infection into the deeper tissues with subsequent complications. Antibiotics, when indicated and in solution, can be applied by this technique. Following the first treatment, medication may be performed preceding each sub-gingival curettage operation.

The routine practice of using antibiotics in periodontal therapy does not appear to be justified because in doing so their efficacy for more serious ailments may be decidedly impaired.[11] We have at hand various drugs whose value and effectiveness have been proved in the past. The only situations that should justify their use are when a patient is suffering from a valvular heart condition or when other drugs have failed to accomplish their purpose.

USE OF COPPER SULPHATE

A valuable drug in periodontal therapy is copper sulphate. Its application is greatly facilitated by incorporating finely ground crystals of this drug in zinc-oxide-eugenol cement as suggested by Dr. A. W. Ward of San Francisco. It can readily be applied when mixed in this form to a medium consistency then placed in a celluloid cement tube and squeezed into the interproximal spaces where it can be locked by pressing with the thumb and the first finger on both buccal and lingual surfaces thereby forming a small button. It should be allowed to remain in position for several days. The The fine crystals dissolve slowly the crevices or pockets to be continuously medicated. Copper sulphate possesses strong antiseptic properties, which act rapidly and thoroughly and is also a spirocheticide thus adding to its value in periodontal therapy. Appleton,[12] in discussing micro-ogranisms of advanced periodontal disease, lists a number of references showing that spirochetes, either with or without fusiform bacilli, have been recognized as prominent members of the flora of periodontal lesions.

When adequately performed, sub-gingival curettage will cause inflammation and infection, in many cases, to subside completely. It should be preceded and accompanied by medication in order to arrest the bacterial flora of the pockets. The principal reason which makes further treatment necessary, where detachment and alveolar resorption are present, is the temporary nature of these results which are nullified by the eventual reinfection of the pockets. It is a fallacy, however, to believe that when eradication methods are employed sub-gingival curettage can be dispensed with. This procedure remains a vital adjunct to the various methods of periodontal treatment as it affords the only means to render the denuded cemental areas free of calcareous deposits, embedded ends of detached connective tissue fibers, necrotic cementum and leave a surgically clean and smooth surface. The curette and sickle type of scaling instruments will satisfactorily meet the needs of most cases for this purpose. The practice of performing sub-gingival curettage, prior to eradication methods rather than after, also permits the growth of new tissue adjacent to smooth and aseptic cemental surfaces in contrast regenerating in the presence of irritating elements.

Teeth exhibiting any degree of mobility, but whose prognosis is favorable, should be relieved of occlusal, lateral and protrusive stresses and interferences through selective grinding. Any loose anterior teeth should then be ligated. Mobility, in the presence of traumatic forces, becomes an aggravating factor in inflammatory changes. Final equilibration may be done following final treatment and when restorative work has been completed.

Where etiological factors are associated with systemic diseases or disorders such as vitamin deficiencies, blood dyscrasias and other constitutional conditions, local treatment alone may prove insufficient to overcome inflammatory changes. In all periodontal cases an evaluation of the systemic factors is absolutely necessary and their

correction is a medical problem that must not be overlooked.

Upon completion of all preliminary measures, for the purpose of relieving inflammatory and infective changes, the gingival tissues should present a firm and rigid texture. In this favorable condition they should be in a state of readiness for any of the pocket eradication procedures.

The surgical dressing, applied following the removal of the pockets, should remain solidly in position the desired length of time without being constantly dislodged and replaced. Failure to do so adds unnecessary discomfort to the patient besides mitigating against the normal regeneration of the soft tissue. The addition of an equal amount of pure liquefied phenol to the liquid component of the cement will produce a resilient, smoother and stronger dressing whose setting point will be sufficiently retarded to permit a heavier mixture and more convenient manipulation. The phenol also increases the sedative properties of the dressing while completely losing its coagulating and escharotic actions in the presence of the eugenol constituent of the liquid. The essayist wishes to acknowledge the suggestion of the use of phenol in this capacity to Dr. Paul J. Boyens of San Francisco. He has used it for many years and has gradually increased its proportion to the prsent amount without any undue effects.

In high strung patients where their nervous condition may influence abnormal post-operative pain and discomfort, the addition of anesthesin (benzocaine) in the proportion of 1-10 to the powder of the dressing will be found to help materially in the control of these symptoms by the analgesic action of this drug. Tannic acid powder in the amount of 1-3 parts of the powder will aid in checking and controlling post-operative bleeding.

The type of periodontal disease which generally responds favorably to pocket eradication methods, is the horizontal atrophy, such as in periodontitis, where the alveolar crest has resorbed uniformly and parallel with the surface of the process. In such cases the new epithelized connective tissue will regenerate to cover the contour of the bone and produce gingival culci of shallow depth. The vertical type of atrophy with intra-alveolar pockets, such as in periodontosis, presents a more difficult problem with either the mesial or distal side of the pocket higher than the other in the interproximal space. In this type of atrophy there is a possibility of the return of the pocket following treatment. This resurgence may be due to the natural tendency of the new tissue to regenerate, across the interproximal space, to the highest level of the alveolar border which may act as a matrix towards this development. Another factor which may contribute to this undesired result, is the possibility of leaving vestiges of inflamed connective tissue at the base of the intra-alveolar pocket and which act as an irritant to stimulate the formation of the superfluous tissue. Evidence to support this theory is given by the results obtained when such a pocket is re-treated with a suitable escharotic which, in its action, will destroy all remnants of the pocket entity. This will be followed by the regeneration of new epithelized connective tissue adhering closely to the irregular contour of the alveolar bone.

The return of the pocket, in either horizontal or vertical atrophies and immediately following treatment or at a later date, may be effectively controlled by chemical means. This expedient has psychological as well as therapeutic advantages. A highly satisfactory drug that fully meets the clinical requirements is sodium hydroxide and calcium oxide as advocated by Dr. A. W. Ward. In the use of this quick acting chemical combination, extreme caution must be employed to protect all surrounding unaffected areas from any contact with it by blocking off with cotton rolls and the use of the saliva ejector. The affected areas are carefully dried and the chemical may be applied with point of a ball burnisher not made of aluminum. The paste

should not be applied in the pocket but carefully outlined on the external surface of the tissue to be destroyed and left in position for not more than three minutes. The amount of the material should not exceed in thickness the flat end of a wooden toothpick. A burning sensation will be produced which generally will not be sufficiently severe to require an anesthetic. The chemical is neutralized by the repeated application of acetic acid (ordinary vinegar strength) on small pledgets of cotton. The eschar formed should be protected for about five days by the use of cement surgical dressing. Upon its removal the eschar should slough away. Caution should always be taken not to allow the chemical to come in contact with exposed bone.

The practice of porte-polishing and taping with an orange wood stick and a suitable abrasive appears to be a forgotten procedure. The only plausible reason for this would seem to be the present vogue of taking short cuts in order to save time and expedite matters. The fact remains that no substitute has yet been devised to replace this method of rendering those portions of both enamel and cementum, susceptible of being areas of irritation to the investing tissues, highly polished and free from irritants for a maximum duration. The use of these procedures to polish subgingival spaces is indicated upon completion of all forms of periodontal therapy and regular prophylactic check-ups.

SUMMARY

1. The approach to periodontal pocket therapies should be preceded by adequate measures to arrest inflammation and infection of the periodontal tissues.

2. The purpose of these measures is to reduce bleeding and post-operative discomfort; to avoid the return of the pockets; to shorten the healing period and prevent bacteremia with systemic consequences.

3. These pre-requisite measures are: medication, sub-gingival curettage and the relief of traumatic stresses and interferences especially on loose teeth.

4. The addition of certain ingredients to the surgical dressing may enhance its utility as regards to its stability in position, its sedative and hemostatic properties.

5. The use of sodium hydroxide and calcium oxide, with proper precautions, may be indicated in advanced intra-osseous lesions and in resurgent pockets in both periodontitis and periodontosis.

6. Porte-polishing and taping should be part of routine practice following all types of periodontal treatments and prophylactic check-ups.

BIBLIOGRAPHY

1. Richards, J. H. Bacteremia following irritation of foci of infection. J.A.M.A. **99**:1496 (Oct.) 1932.

2. Hopkins, J. A. Streptococcus viridans: bacteremia following extraction of teeth. J.A.D.A. **26**: 2002 (Dec.) 1939.

3. Murray, M., and Moosnick, F. Incidence of bacteremia in patients with dental diseases. J. Lab. & Clin. Med. **26**:801 (Feb.) 1941.

4. Fish, E. Wilfred. Local and remote sequelae of infection in the paradontal sulcus. Proc. Roy. Soc. Med. Sec. Odont. **30**:1149 (July) 1937.

5. Mitchell, F. David, and Helman, Z. Edith. The role of periodontal foci of infection in systemic disease: an evaluation of the literature, **46**:53 (Jan.) 1953.

6. Burket, W. Lester. Oral Medicine, Philadelphia: J. B. Lippincott Co., p. 532, 1940.

7. Easlick, A. Kenneth. An evaluation of the effect of dental foci of infection on health. J.A.D.A. (June) 1951.

8. Wesson, B. Miley. Excerpts from—Focal Infection: Its Role in General Health. Contact Point **31**:144 (Feb.) 1953.

9. Kolmer, A. John. Focal infection in relation to health and disease. J.A.D.A. **45**:139 (August) 1952.

10. Vastine, A. B. Research in sub-gingival therapy. J.A.D.A. **17**:507-11 (March) 1930.

11. Answers to queries in editorial. J.A.D.A. **46**:217 (Feb.) 1953.

12. Appleton, J. L. T. Bacterial infection with special reference to dental practice, ed. 3 Philadelphia, Lea & Febiger, 1950, p. 524.

Tissue Reactions Around Artificial Crowns

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EARLIER INVESTIGATIONS

IN the literature concerning crown and bridge work, statements have been made about tissue-phil and non-tissue-phil materials used for artificial crowns, but the presumed differences have been founded only on clinical observations. Histological investigations, which should be considered absolutely necessary for such evaluations, seem to be absent. *Immenkamp* (1932) and *Dewey & Zugsmith* (1933), however, touch upon the subject as they deal with the healing of the tissue around artificial root tips. As to the epithelium and its relation to the tips, it is difficult to advance any opinion, as the epithelium had been torn when the tips were taken out. In a monograph on the gingival pocket *Waerhaug* (1952) observed that apparently normal epithelial cells could adhere to the surface of artificial crowns which had been situated below the gingival margin for some time, and that in the same region there could be found a horn stained membrane similar to the dental cuticle in the region of the so-called "epithelial attachment" (*Gottlieb*, 1921:1). On the basis of this and other findings the conclusion was drawn that the connection between the "epithelial attachment" and the tooth does not consist in a real growing together, but in an adhesion, and that the bottom of the clinical pocket is to be found at the deepest border of the "epithelial attachment" and not, as was earlier presumed, at its outer border. For didactic reasons it was suggested that the term "epithelial attachment" be replaced by the term epithelial cuff as the latter seems to be more suggestive of the actual condition in the gingival pocket. Furthermore it was presumed possible to attain as ideal conditions around an artificial crown as around a natural tooth. This conclusion was supported by *Waerhaug & Steen* (1952) who found that pockets around well fitting artificial crowns under favorable conditions could be sterile.

STATEMENT OF PROBLEMS

The purpose of the present investigation was to study the reaction of the gingiva to artificial crowns situated below the gingival margin. Do they lead to a chronic inflammation or is it possible to obtain conditions similar to those around natural teeth? Will an artificial crown reaching the bottom of the original pocket cause a deepening of the latter, or is it possible to maintain a status quo? What significance has a good or deficient fitting for the result? These are important problems, the elucidation of which seems to be of great significance for clinicians.

OWN INVESTIGATIONS

Material and Method.

Two dogs with absolutely healthy gingiva were used for the investigation, dog No. 5, age at death 39 months and dog No. 8, age at death 19 months. On dog No. 5 were used $\underline{4\ 3\ 2}|\underline{2\ 3\ 4}$ and the observation period varied between 80 and 84 days. On dog No. 8 were used $\underline{7\ 6\ 3\ 1}|\underline{6\ 7}$ / $\underline{8\ 7\ 6}|\underline{6\ 7}$ and the observation period varied between 26 and 55 days. The crowns were made of ordinary thermocuring acrylic resin (Stellon C). The preparation and the application of the crowns were carried out under Nembutal narcosis. There was no deepening of the pocket beneath the cemento-enamel junction around any of the teeth used, and attempts were made to avoid deepening also during the preparation. Endeavors were made to prepare shoulders or bevels. Impressions were taken with modeling compound and packed with amalgam. Temporary tin caps were fastened on the stumps with phosphate cement in order to keep the gingiva aside. Some of

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these were chewed off, however, before the crowns were ready. On closer inspection of the acrylic crowns under the stereomicroscope it appeared that the fitting to the amalgam dies was not always perfect. They were nevertheless put in, as it would be of interest to study the tissue reaction both to perfectly and imperfectly adapted crowns. The crowns were fastened with De Trey's crown and bridge cement. In those cases where the temporary tin caps had fallen off, the gingiva was kept aside as carefully as possible during the cementation, even though such a proceeding must be considered very unsatisfactory. All attempts to press the gingiva aside by means of new temporary tin caps appeared to be unsuccessful, however, as these were always chewed off before the next "sitting." The cement having set, the excess was removed. In the course of the observation period five of the crowns fell off. This was probably due to the elastic acrylic crowns not being able to bear the extreme stress to which they are exposed in the mouth of a dog.

After the animals had been sacrificed, the jaws were carefully taken out and fixed in 10% formalin according to the principles drafted by *Waerhaug* (1952, pp. 29-33). After three days of fixation, the whole jaws were transferred directly into chloroform in order to dissolve the acrylic crowns. By dissolving these instead of taking them out, it was presumed that the relation between the soft tissue and the crowns would be analogous to that which exists in natural teeth, when the enamel has been dissolved in acid. The jaws were left in chloroform until the last remnant of acrylic resin had been dissolved. This was controlled under the stereomicroscope. Especially in dog No. 5 it was observed that a relatively large amount of cement had remained on the outside of the crowns near their cervical margin. All the acrylic resin having been removed, the jaws were transferred into 5% nitric acid, in which solution they were left until it was determined by X-ray control that they were completely decalcified. The jaws were then cut into pieces of proper size for sectioning. Special care was taken not to move the gingiva in relation to the crowns until the decalcification was completed, lest the tissue should be torn. The pieces of jaw were embedded in paraffin and sectioned bucco-lingually. There are 90 to 150 sections from each tooth. The sections were stained in hematoxylin-eosin and in Azan.

In addition to the animal material, there are sections from the gingiva around ten porcelain jacket crowns and five gold crowns obtained from autopsy material. In these cases the crowns had to be taken out after decalcification, for which reason the tissue resting upon the crowns was often more or less torn. The specimens of this human material were sectioned mesio-distally, and were embedded some in paraffin, some in celloidin.

Clinical observations in the dogs during the observation period.

At each new treatment the gingiva was examined around the crowns already cemented. After eight days or more the gingiva proved to be clinically "healthy" around all the crowns, which means that there was no noticeable difference between these and teeth on which no operation had been performed. The reservation must be made, however, that in the gingiva of the dogs there was some pigment which made it difficult to determine whether there was any hyperemia. In a few cases, however, small quantities of pus could be forced out of the pocket by hard pressure.

Observations.

Experimental dog material. As the reaction varied over a wide range, efforts have been made to select typical cases for the illustrations.

Fig. 1 shows the poorest result observed. In the general view the junction between the enamel and the acrylic crown can be seen at J. There is a heavy infiltration of leukocytes in the connective tissue and a thickening of the epithelium. At the higher

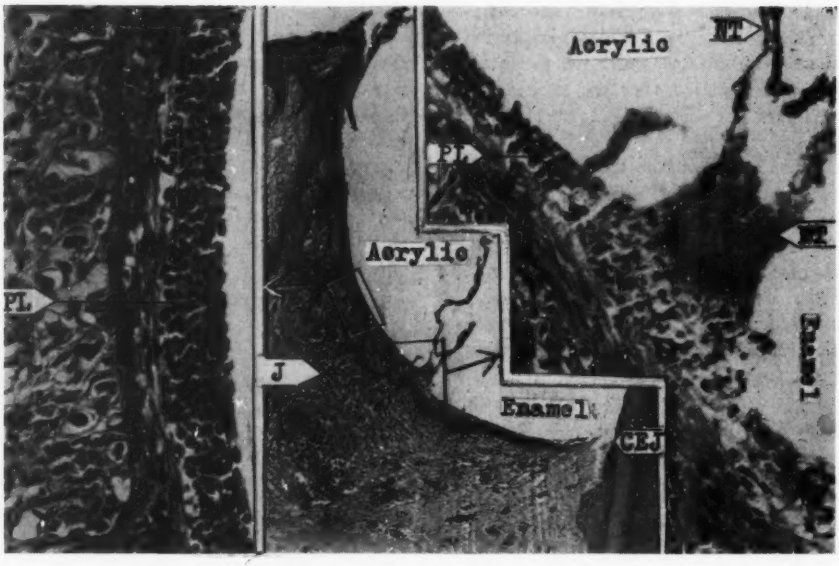

Fig. 1. Dog 8, $\overline{8}$|. Observation period 43 days. Section from tissue around acrylic crown (dissolved in chloroform before sectioning). Pocket above junction, J, filled with polymorphonuclear leukocytes, PL. Space between acrylic crown and enamel filled with crown and bridge cement, and necrotic tissue, NT. Heavy infiltration in connective tissue with thickening of epithelium. No deepening of original pocket.

magnification to the right it is seen that the crevice between the acrylic crown and the enamel is filled with necrotic tissue, NT, which also stretches between the acrylic crown and remnants of the crown and bridge cement. Between the acrylic crown

ABBREVIATIONS USED IN ALL ILLUSTRATIONS

Acr.—Acrylic:	Space in which acrylic crown was in contact with soft tissue before being dissolved in chloroform.
Art	Artifact.
BP	Bottom of clinical pocket.
Ca	Calculus.
CBC	Crown and bridge cement.
CEJ	Cemento-enamel junction.
"CU"	Membrane resembling primary cuticle on surface of acrylic or jacket crowns.
D	Degenerated epithelial cells.
EM—Enamel:	Space occupied by enamel before being disolved in acid.
"HCu"	Membrane resembling secondary (hornified) cuticle on surface of acrylic crown.
I	Infiltration in connective tissue.
J	Junction between acrylic crown and enamel or cementum.
Jacket Crown:	Space occupied by jacket crown before removal, after decalcification.
NT	Necrotic tissue.
P	Plaque.
Reat	Reattachment.
PL	Polymorphonuclear leukocytes.

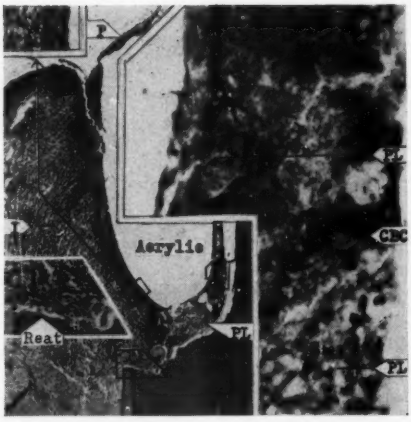

Fig. 2. Dog 5, 3|. Observation period 84 days. Section from tissue around acrylic crown. Space between acrylic crown and tooth filled with polymorphonuclear leukocytes, PL. Between pocket epithelium and acrylic crown accumulations of polymorphonuclear leukocytes. Heavy infiltration in connective tissue.

and the epithelium there are several layers of polymorphonuclear leukocytes, PL. The border towards the crown is relatively distinct, but no formation of a continuous membrane is observed. There is no deepening of the pocket beneath the cemento-enamel junction, CEJ, but a thickening of the cementum below it.

Fig. 2 shows similar conditions. The border towards the acrylic crown is distinct, but also here many polymorphonuclear leukocytes are found adjacent to the crown. The preparation ends below the cemento-enamel junction, and the adaptation of the crown has been imperfect. During the cementation the space between the crown and the tooth has not been completely filled with cement, and for the moment the exterior part of it is filled with polymorphonuclear leukocytes, PL. At the higher magnification to the right such cells may be seen surrounded by necrotic tissue inside lacunae in the cement, CBC. At the outer part of the shoulder, reattachment is attained, Reat.

Fig. 3 represents one of the cases where, after the dissolving of the acrylic crown, large quantities of cement were observed

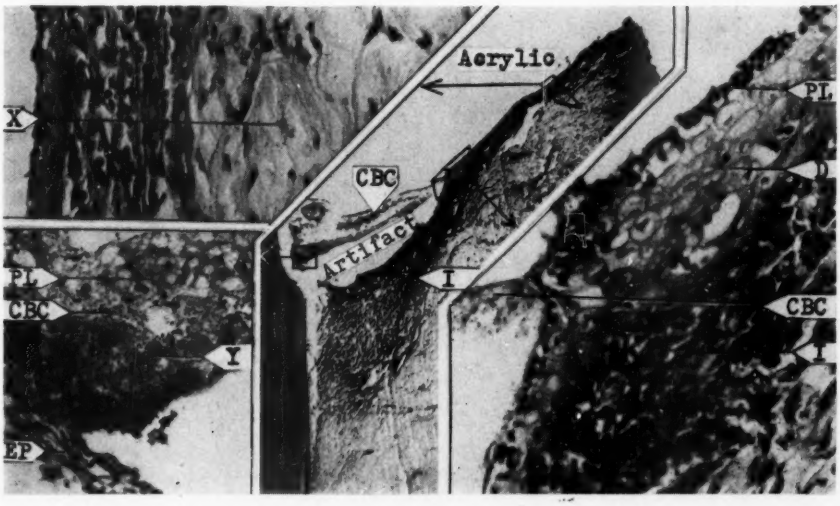

Fig. 3. Dog 5, |4. Observation period 84 days. Section from tissue around acrylic crown on which were left large quantities of crown and bridge cement, CBC. Heavy infiltration in connective tissue most pronounced corresponding to excess cement. At the bottom to the left, section from excess cement with degenerated and necrotic tissue in small cavities inside cement. No deepening of pocket below prepartion.

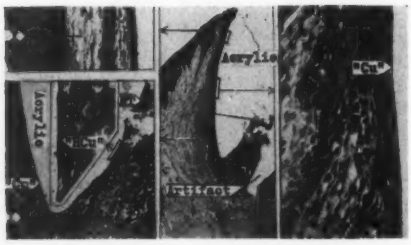

Fig. 4. Dog 8, $\overline{6}|$. Observation period 55 days. Section from tissue around acrylic crown. Space between acrylic crown and tooth surface filled with necrotic tissue, polymorphonuclear leukocytes and flap of granulation tissue. Pocket epithelium joining closely to membrane resembling primary cuticle, "Cu". "HCu", cuticle-like membrane taking horn stain and resembling secondary cuticle. Only slight deepening of pocket under deepest point of preparation.

adjacent to the soft tissue. This may be seen in the section at CBC. In the subjacent connective tissue there is a heavy infiltration of leukocytes, I, which decreases both along the root and towards the gingival margin, X. The higher magnification at the bottom to the left shows a section from the cement. Small lacunae in this contain polymorphonuclear leukocytes, PL, and closer to the surface lies necrotic tissue, Y, which assumed a reddish color in hematoxylin-eosin. Towards the cement and, though to a smaller degree, towards the crown, the epithelium is degenerated and bordered outwardly by polymorphonuclear leukocytes. There is no deepening of the pocket below the deepest point of the preparation.

Also Fig. 4 shows an imperfectly adapted crown, a lobe of gingiva having remained between the crown and the preparation. There is a heavy infiltration of polymorphonuclear leukocytes in the subepithelial connective tissue (see higher magnification at the bottom to the left). Even here, there are small quantities of necrotic tissue, NT, and a bit further down on the surface of the acrylic crown there is a membrane, "HCu," which both in hematoxylin-eosin and in Azan assumed the typical horn stain. It proved to be epithelial cells which have been in contact with the acrylic crown all the way to the gingival margin. The epithelial cells show signs of degeneration, and are outwardly bounded by a thin membrane, "Cu." Nearer to the gingival margin the infiltration is insignificant, X.

In Fig. 5 the fitting has been excellent.

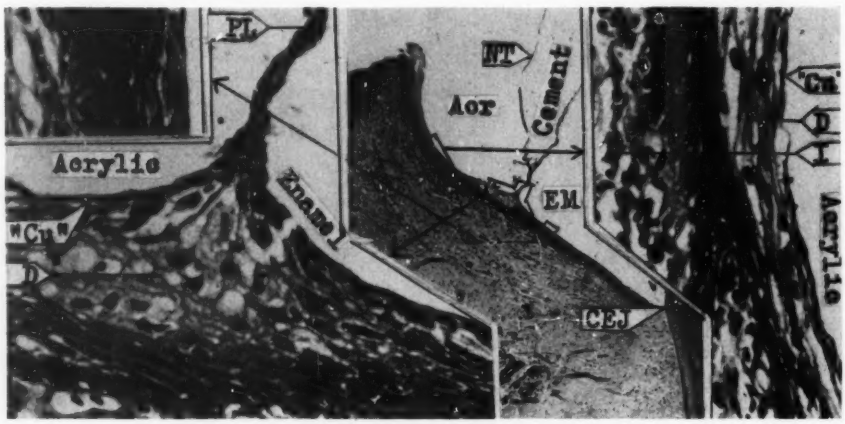

Fig. 5. Dog 8, $|\underline{6}$. Observation period 43 days. Section from tissue around acrylic crown. Space between acrylic crown and enamel filled with polymorphonuclear leukocytes, necrotic tissue and cement. Pocket epithelium degenerated, but joining closely to membrane on surface of acrylic crown to gingival margin. Infiltration in connective tissue, mostly above junction between acrylic crown and enamel. No deepening of original pocket.

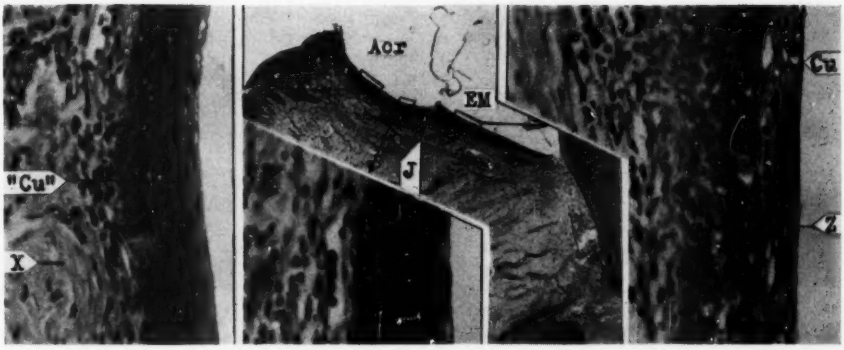

Fig. 6. Section taken from opposite part of tooth surface shown in Fig. 5. Comparatively normal epithelial cuff joining closely to cuticle-like membrane, "Cu", on surface of acrylic crown (left). Epithelial cuff on enamel bordered by distinct cuticle, Cu, which disappears further down, Z (right).

Thanks to a very cautious histological preparation, also most of the cement, with which the crown was fastened, has been preserved. At the higher magnification to the left the junction between the acrylic crown and the enamel may be seen. In the microscopic crevice between them there are a number of polymorphonuclear leukocytes, PL. Corresponding to the junction the epithelial cells are degenerated, D, and towards the acrylic crown they are bordered by a thin membrane, "Cu." Similar conditions are also to be found closer to the gingival margin (see higher magnification to the right). There is an infiltration of polymorphonuclear leukocytes in the connective tissue between the deepest point of the crown and the gingival margin.

Fig. 6 shows the opposite part of the same tooth surface as in Fig. 5. Here an approximately normal epithelial cuff borders upon a thin membrane, "Cu," on the surface of the acrylic crown. To the right will be seen the epithelial cuff adjacent to the enamel. At the top there is a marked cuticle, Cu, which is reduced in thickness towards the cemento-enamel junction and which is but vaguely perceptible at Z.

Fig. 7 shows a preparation that has been carried past the cemento-enamel junction and the crown does not cover the whole shoulder. The adaptation has been very good in as much as no space is to be seen between the tooth and the crown. Between the edge of the shoulder and the crown there is a connective tissue reattachment with cementum formation, Reat. In

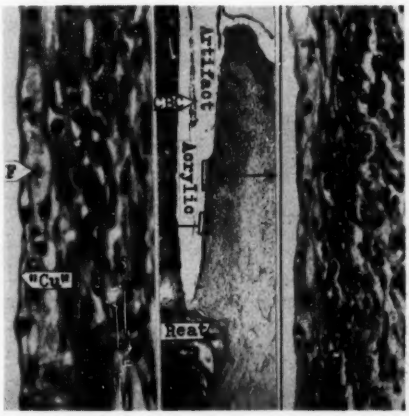

Fig. 7. Dog 5, 3. Observation period 84 days. Section from tissue around acrylic crown. Reattachment in part of preparation just beneath lower margin of crown. Crown and bridge cement, CBC, left behind on crown just below gingival margin, and corresponding to this heavy infiltration in connective tissue. Infiltration missing at lower margin of crown. Pocket epithelium in close contact with membrane on surface of acrylic crown. Pocket epithelium degenerated.

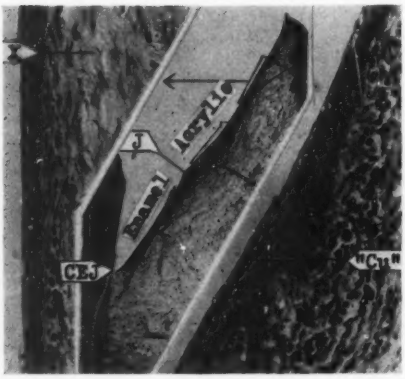

Fig. 8. Dog 8, $\overline{6|}$. Observation period 55 days. Section from tissue around acrylic crown. Epithelium joining closely to membrane, "Cu", on acrylic crown up to gingival margin. Only slight thickening of epithelium. Some infiltration in connective tissue corresponding to junction between acrylic crown and enamel, J. Infiltration practically disappeared at X.

the upper half of the pocket some cement remains on the surface of the crown, CBC, and corresponding to this may be seen a heavy leukocyte infiltration in the subepithelial connective tissue. This infiltration decreases in intensity towards the lower edge of the crown. Also the epithelium is infiltrated by polymorphonuclear leukocytes, soaked by fluid, F, and bordered outwardly by a thin membrane, "Cu."

The conditions in the case shown in Fig. 8 are more favorable. Corresponding to the junction between the acrylic crown and the enamel, the pocket epithelium is slightly thickened, but the cells show few signs of degeneration and are outwardly bordered by a thin membrane, "Cu," (see higher magnification to the right). In the subjacent connective tissue there is a moderate infiltration of polymorphonuclear leukocytes. The infiltration decreases in intensity towards the gingival margin and has almost disappeared at X (see higher magnification to the left). The pocket epithelium is here of normal thickness and adapts itself closely to a thin membrane all the way to the uppermost edge of the gingiva. Beneath the cemento-enamel junction a considerable thickening of the cementum may be found, but there is no deepning of the pocket.

In Fig. 9 the conditions are much the same. The preparation has in this case been carried below the cemento-enamel junction, and the lower edge of the acrylic crown has been situated at J. The pocket is somewhat deepened below the shoulder, and small quantities of cement have been pressed down into the tissue, CBC. A comparatively normal pocket epithelium has been in contact with the acrylic crown all the way to the gingival margin. Exteriorly it is bordered by a membrane, "Cu." The infiltration in the connective tissue is most pronounced in the region around J and decreases evenly towards the gingival margin.

Autopsy material. The two illustrations selected from the autopsy material (Figs. 10 and 11) are taken from a 45 year old healthy man who died by accident. Available information showed that ten porcelain jacket crowns had been put in at least two years before death. The two above mentioned illustrations are representative for this individual and also for the rest of the material (five gold crowns).

Fig. 10. Adjacent to the porcelain jacket crown the pocket epithelium is of rather normal appearance compared to what it is on the opposite side, where it has been in contact with the calculus, Ca. Between the epithelium and the space left by the crown there is an accumulation of leukocytes, PL. The formation of a membrane is not apparent, but the border is distinct. Below the deepest point of the preparation a normal epithelial cuff stretches 0.4 mm along the cementum. For the sake of comparison it should be observed that the epithelial cuff on the opposite side stretches 1.8 mm below the deepest point of the calculus, and that the alveolar bone is correspondingly resorbed.

Fig. 11. The pocket epithelium adjacent to the left jacket crown shows moderate signs of irritation in the form of thickening and epithelial pegs, but is otherwise of relatively normal appearance. It is bordered

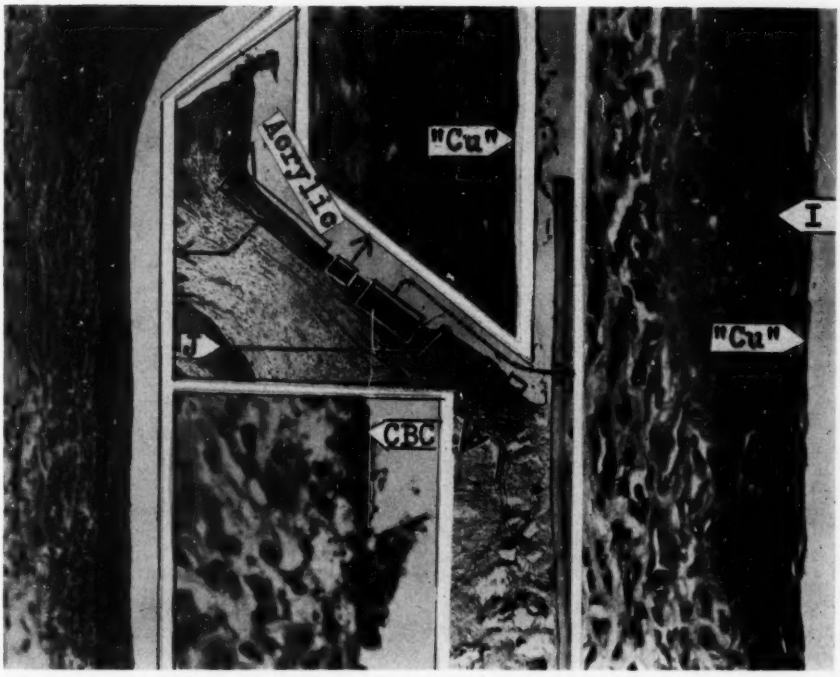

Fig. 9. Dog 8, 1. Observation period 26 days. Section from tissue around acrylic crown. Pocket epithelium joining closely to acrylic crown to gingival margin and resembling normal cuff epithelium ("epithelial attachment"). Crown and bridge cement pressed into pocket beneath lower margin of crown. Infiltration in connective tissue decreasing towards gingival margin.

by a thin membrane, "Cu." Between the epithelial cells there are a number of polymorphonuclear leukocytes, PL, and furthest down lies a black mass, CBC, consisting of cement grains (see higher magnification to the left). The infiltration in the connective tissue is most pronounced corresponding to the junction between the jacket crown and the tooth, I. At the bottom to the right has been inserted a higher magnification of a fragment of cement which has lain in contact with the epithelium. Small lacunae in the cement contain degenerated and necrotic cells. On the right tooth, calculus, Ca, has been formed beneath the jacket crown and the epithelium shows considerably greater signs of irritation. The round cell infiltration in the connective tissue is most pronounced corresponding to the deepest part of the calculus. The bottom of the pocket, BP, is here situated 1.7 mm beneath the deepest part of the crown, while on the other side the distance is only 0.7 mm.

DISCUSSION

The experiments in dogs give an idea of the great variation in tissue reactions which can be expected around artificial crowns. The heaviest inflammation was found in Figs. 1, 2, 3, and 4, where, during the application of the crowns, there was either an excess of cement (Fig. 3), or where the space between the crown and the cement had not been completely filled out (Figs. 1, 2, and 4). In the last three cases there is necrotic tissue furthest down in the

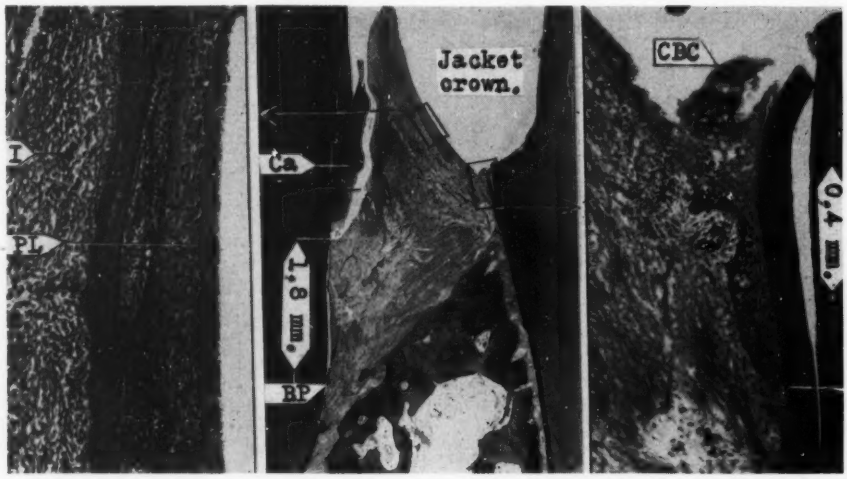

Fig. 10. Celloidin section from 45 year old man on whom porcelain jacket crown was applied more than two years earlier. Crown removed after decalcification. Pocket epithelium adjacent to porcelain jacket crown shows relatively slight signs of irritation compared to pocket epithelium adjacent to calculus. Infiltration in connective tissue most pronounced corresponding to lower part of porcelain jacket crown.

crevice, and this tissue is bounded by a zone of polymorphonuclear leukocytes. Whether this necrotic tissue also contains bacteria, cannot be maintained with certainty, since no cultures were made, but the histological picture, especially in Fig. 1, resembles the necrotic deposits which form the initial stage of calculus, and this kind of deposit is always infected according to investigations by *Waerhaug & Steen* (1952). Therefore, it is probably the bacteria that, in Fig. 1, have caused the conspicuously heavy irritation. In Figs. 1, 2, and 3 the emigration of leukocytes has been so extensive that a great number of them during the excretion have remained between the epithelium and the acrylic crown, and it must have been pus cells of this kind that were observed in a few cases at the clinical investigation. In Fig. 4 it is mostly epithelial cells that have lain close to the crown, and these are outwardly bordered by a thin membrane, "Cu," which in the section corresponds to the terminal membrane that in decalcified specimens is found adjacent to the enamel and which has been called the primary enamel cuticle, according to *Gottlieb* (1921:2). The same close contact between the pocket epithelium and such a "cuticle" is also found in Figs. 5, 6, 7, 8, and 9. In some cases the epithelial cells are more or less degenerated (Figs. 4, 5, and 7), but in others they correspond remarkably well to what is found in a normal "epithelial attachment" (Figs. 6, 8, and 9). Figs. 4-9 show that the histological technique used has been satisfactory. During the fixation the epithelium has adhered to the acrylic crowns, and by means of dissolving these in chloroform instead of extracting them it has been possible to save the tissue from being torn. Thus, the conditions on the border-line between the dissolved acrylic crown and the epithelium should be directly comparable with those between the enamel and the epithelium when the former has been dissolved in acid. The present investigation accordingly supports the assertion that findings of epithelial cells in direct contact with the enamel, or possibly an enamel cuticle, cannot be taken as evidence of an organic

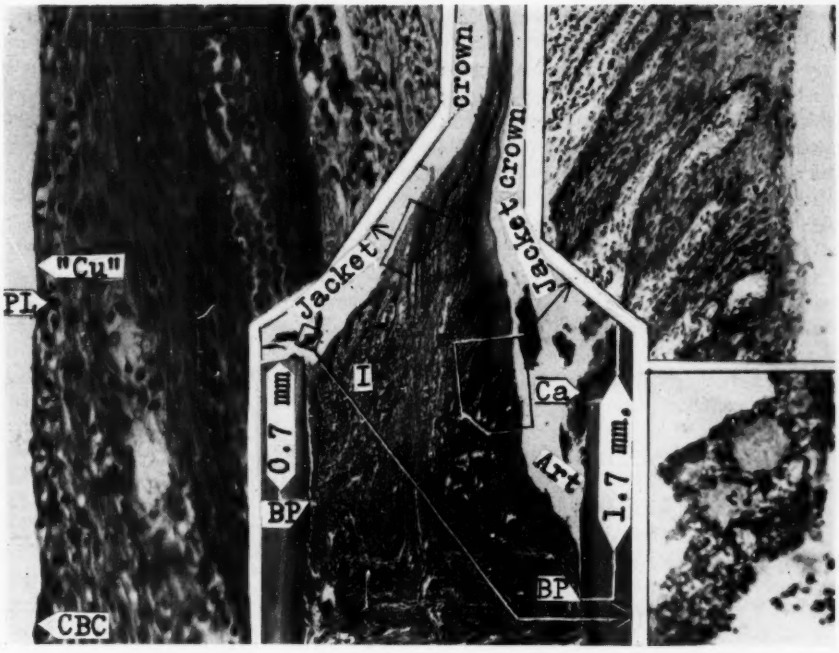

Fig. 11. From the same individual as foregoing. On left side well fitting porcelain jacket crown. Corresponding to shoulder small quantities of crown and bridge cement remain in which are seen necrotic and degenerate cells. Pocket epithelium joining closely to cuticle-like membrane, "Cu", on surface of porcelain jacket crown. Infiltration, I, in connective tissue most pronounced corresponding to lower part of porcelain jacket crown. On right side calculus, Ca, formed below deeper part of porcelain jacket crown. Infiltration in epithelium and connective tissue more pronounced on this side.

connection having existed between the two tissues (*Waerhaug*, 1952). The theory about the "epithelial attachment" (*Gottlieb*, 1921:1) is solely based on observations of the close contact between the epithelium and the tooth surface in histological sections. Since it is possible to make quite similar observations in cases where the tooth surface has been replaced by foreign bodies, such as acrylic or porcelain crowns, it seems to be highly justified to revise this theory.

According to the general conception which is for the most part based on histological investigations by *Gottlieb* (1921:2), there exists on the surface of the tooth, when it breaks through the gingiva, a primary enamel cuticle which is considered to be calcified and to belong to the enamel. In Figs. 5, 6, 8, and 9, in the region where the enamel has lain, a cuticle corresponding to this description may be observed. In the same illustrations, however, there is a similar "cuticle" adjacent to the acrylic crowns which, as far as could be determined in an ordinary microscope, appeared to be absolutely identical with the former. It seems, therefore, as though the so-called primary cuticle is not specific for the enamel. Until conclusive evidence has been given, it ought to be regarded as the exterior, not necessarily calcified layer of the epithelial cuff. It may possibly represent a coherent layer of cell membranes, it may be cell remnants

or a fibrin film. One must also take into consideration that optic phenomena may play a rôle. In Fig. 4 in a limited region a relatively thick membrane may be seen which assumed a distinct horn stain in hematoxylin-eosin and in Azan ("HCu"), and similar observations were made on five other crowns. Analogous findings have been made previously by *Waerhaug* (1952) on the surface of excess cement left behind beneath the gingival margin, and on root filling material. The same author also found a "horn membrane" on the surface of artificial crowns that had reached below the gingival margin. The observation made in Fig. 4 confirms these findings. As the "horn membrane" in Fig. 4 is situated in a highly inflamed region, it is most likely a disintegration product. If also the primary cuticle is presumed to be a product of the epithelial cuff, the question arises whether there is any qualitative difference in reality between the primary and the secondary cuticle. Thus, it is possible that the difference is only quantitative—that in the thin layers the membrane does not take sufficient stain to be perceptible in the microscope, while in thicker layers it will be visible. At the higher magnification to the right in Fig. 6 there is at the top what could be called a well developed primary cuticle, Cu, which becomes thinner and almost disappears at Z. It seems most probable that this variation in the thickness of the cuticle is due to its upper part having been situated closer to J where the irritation has been greatest, and that, as a consequence of this, remnants of necrotic cells have settled on the enamel surface.

After these observations it appears to be necessary to carry out further investigations on the primary and secondary cuticle and determine whether there is any difference between them.

It may be of interest to notice that in Figs. 2 and 7 connective tissue reattachment is attained in the part of the shoulder which was not covered by the crown. In Fig. 2 the strength of the attachment is rather doubtful, there being no evident cementum formation with periodontal fibers. In Fig. 7, however, a thin layer of cementum is formed to which Sharpey's fibers are attached, and there is a very slight inflammatory reaction in the connective tissue corresponding to the deepest part of the crown. There is reason to believe, therefore, that this reattachment would have persisted. This investigation shows, accordingly, that a well adapted crown which ends precisely at the bottom of the pocket need not cause an irritation leading to a further deepening of the pocket in dogs.

In Fig. 9 such a deepening has developed, however, and the cause is probably irritation from excess of cement pressed into the connective tissue between the crown and the bottom of the pocket. During the preparation or the impression taking a deepening of the pocket has most likely been induced beneath the shoulder, and this artificial pocket has, at the cementing of the crown, been filled with cement which has not been completely removed later on. The possibilities of reattachment are thus excluded. In a way it is remarkable not to find *more* loose fragments of cement left behind in the pockets after the cementation. Fortunately, though, gingival pockets have an exceptional capacity of ridding themselves of foreign bodies. Thus, *Waerhaug* (1952) found that India ink, inserted into healthy pockets, had disappeared after about three days, and *Waerhaug & Steen* (1952) found that bacteria were likewise eliminated. Grains of India ink and bacteria are small enough to be attacked directly by leukocytes. The separate crystal of cement, however, is infinitely much larger, and it is still worse when whole fragments of it are concerned. Therefore, it is probable that the secretion of pus is of importance here. On account of the irritation caused by the fragments of cement, these are surrounded by pus in the pocket, and by a pressure on the gingiva the pus will be squeezed out, and with it the fragments of cement.

An examination under the stereomicroscope, after the acrylic crowns had been

dissolved in chloroform, showed that in some cases great quantities of cement had been left behind during the cementation, and it is such remnants that may be seen in the section in Figs. 3 and 7 at CBC. It is apparent that the cement has here caused a severe irritation, which can be seen by the cell infiltration in the connective tissue. A similar reaction was also found in all the other cases where excess of cement was left behind. This irritation caused by an excess of cement is hardly more than ought to be expected, and it corresponds to observations made by many clinicians.

WEAK POINT

With the exception of Fig. 7, the most pronounced infiltration is to be found in a region corresponding to the junction between the acrylic crown and the tooth. This junction, therefore, is obviously the weak point of the technique. Here an overhang or a defect may occur, and between the crown and the tooth there will be a crevice of various dimensions practically all around. During the fastening of the crown the intention is to fill out this crevice with cement. This is not always so easy, however, as will be seen in Figs. 1, 2, 4, and 5. Even if the space is not broader than in Fig. 5, it is still sufficient to permit cells and tissue fluid to enter it. These cells will soon degenerate and necrotize, and the disintegration products will ooze out and irritate the surrounding epithelium. In Fig. 5 there are probably no bacteria in the space, since the condition would then be more like that in Fig. 1. But even where the crevice is completely filled with cement, there is a possibility of irritation. Thus, in Figs. 2, 3, and 11 it may be seen that deep inside the cement there are lacunae in which degenerated and necrotic leukocytes have accumulated. Even without the presence of bacteria, there is reason to believe that the necrotic tissue will irritate the tissue with which it comes in contact, and if there really are bacteria present, it seems to be obvious that these can never be destroyed. It has been shown by *Waerhaug & Steen* (1952), that pockets infected with known bacteria are sterile again after 48 hours at most, and that pockets in which subgingival calculus has been present can become sterile if the calculus is removed. The condition for this, however, is a smooth tooth surface without microscopic hollows where the bacteria can take refuge against the attack of the phagocytes. Unfortunately such recesses are frequent in the cement, and if they are once invaded by bacteria, it is hardly probable that these could be eliminated. There is reason to believe, therefore, that the polished acrylic crowns do not themselves cause any significant irritation, but that the inflammations in the above mentioned cases are due to irritation from necrotic tissue remnants and/or bacteria between the crown and the tooth or inside the excess of cement. If the fitting is good, it is not probable that such an insignificant irritation would cause a deepening of the pocket, reattachment having been attained in Fig. 7 all the way to the deepest point of the crown.

From these observations on experimental dog material it should be permissible to draw certain conclusions from the human material shown in Figs. 10 and 11. Thanks to the great caution observed when the crowns were removed, the pocket epithelium has not been seriously torn. In Fig. 11 a thin membrane has even been preserved on the surface which seems to be identical with the one observed on the surface of the acrylic crowns. It is for the most part epithelial cells that lie in contact with this membrane, but among these cells there are also many polymorphonuclear leukocytes which are presumably migrating out of the pocket. This seems to be true also of the small cement crystals lying between the epithelium and the jacket crown. Both in Figs. 10 and 11 the moderate infiltration in the connective tissue inside the crowns is most pronounced at their lower edge. It is natural to presume, therefore, that also in human beings it is the cement and not the porcelain that is the main irritating factor. In the two figures the inflammation process is conspicuously more intense where the tissue has lain in contact with the cal-

culus, than where it has been in direct contact with the jacket crowns, and at the same time the pockets are much deeper. In Fig. 10 it may furthermore be seen that the interdental septum is considerably more resorbed corresponding to the calculus. The irritation exercised by a well fitting jacket crown, accordingly, can by no means be compared with that of calculus. That the jacket crown or the cement cannot cause an irritation leading to a grave deepening of the pocket is also clearly seen in Fig. 10 where the bottom of the pocket is situated only 0.4 mm beneath the deepest point of the excess of cement, CBC. It is hardly probable that the pocket has here been deepened after the crown was fastened, since it must be considered very difficult to prepare and cement a crown closer than 0.4 mm to the bottom of the pocket. There should be quite good possibilities, therefore, of avoiding a destruction of the supporting apparatus of the tooth with a subsequent deepening of the pocket, if the preparation were finished about 0.4 mm above the bottom of the original pocket, and a good adaptation were attained. This conclusion is founded on, among others, the circumstance that on two more crowns from the autopsy material distances as small as 0.4 mm were found, and on an observation made by *Waerhaug* (1952) who found the bottom of the pocket only 0.6 mm beneath the deepest point of a gold crown 10 years after it was put in. It is also supported by the findings made in Fig. 7, where reattachment was attained all the way to the cervical margin of the crown.

According to findings in the autopsy material, it may be assumed that the reactions to artificial crowns are not principally different in dog and man, and that there is no significant difference in the reaction of the tissue to porcelain and acrylic resin. For practitioners the investigation affords rather reliable guidance. On patients with a marked caries disposition, it can often be of vital importance to place the edge of the crown as deep as possible beneath the gingival margin, in order to avoid an exposure of the root when the gingiva recedes. If a good fitting of the crown is secured, it should be safe to make the preparation as near to the bottom of the pocket as possible, without going beyond it. It is hardly possible to get closer than 0.4 mm, and at this distance there are slight chances of a deepening of the pocket.

SUMMARY AND CONCLUSIONS

Dogs. Acrylic crowns which reached below the gingival margin were applied to a total of 17 teeth in two dogs. The observation period varied between 28 and 84 days. After the animals had been sacrificed, the jaws were fixed for three days, whereupon the crowns were dissolved in chloroform, and the jaws decalcified and sectioned. By means of dissolving the acrylic crowns, it was possible to preserve the tissue which, in the section, had the same appearance as after dissolving enamel in acid. The following observations were made:

1. Under favorable conditions the epithelium joined closely to the acrylic crowns in the same way as the epithelial cuff ("epithelial attachment") joins around a natural tooth. The acrylic crown in itself, therefore, does not appear to cause any significant irritation.

2. On the border between the acrylic crown and the epithelial cuff a membrane was observed which, in an ordinary microscope, completely resembled the primary cuticle between the epithelial cuff and the enamel. This finding raises the question whether the so-called primary cuticle belongs to the enamel and whether it is calcified.

3. In some cases a thicker membrane was found between the epithelium and the acrylic crown, and this membrane took horn stain. The question is, therefore, whether the difference between the primary and the secondary cuticle is not quantitative instead of qualitative.

4. Under imperfectly adapted crowns, in the space between the crown and the tooth, necrotic tissue was observed which caused irritation of the epithelium and the connective tissue.

5. Where large quantities of cement were left behind in the pockets, these acted as a strong irritant, but even the small quantities necessary for filling the space between a well fitting crown and the tooth will have some irritating influence, without the subsequent slight inflammation necessarily causing a deepening of the pocket under otherwise favorable conditions. However, a substance with less irritating effect than cement would be desirable for this purpose.

6. In one case where the preparation had passed beyond the bottom of the existing pocket, and where the crown did not completely cover the prepared region, reattachment was attained all the way to the cervical margin of the crown. It is not probable, therefore, that well fitting crowns cause an irritation leading to a deepening of the pocket.

Autopsy material. A histological investigation of the tissue around ten well fitting porcelain jacket crowns and five gold crowns removed after decalcification revealed the following:

7. There seems to be no principal difference between the tissue reaction to artificial crowns in dog and man, nor does the tissue appear to react differently to porcelain, gold, and acrylic resins.

8. A well adapted artificial crown need not cause an irritation leading to a further deepening of the pocket, when the cervical margin of the crown does not come closer than 0.4 mm to the bottom of the pocket.

9. In practice this means that a crown can be prepared as close to the bottom of the existing pocket as is technically possible without severing the periodontal fibers. The requirements for this, however, are a perfect fitting, a thorough removal of all living and necrotic tissue between the tooth and the crown before the latter is cemented, a total filling of the space with cement, and finally a complete removal of the excess cement.

10. If the requirements mentioned under point 9 cannot be fulfilled, an artificial crown should not be placed below the gingival margin.

REFERENCES

Dewey, Kathe and Zugsmith, R.: An experimental study of tissue reactions about porcelain roots. J. Dent. Res. **13**:459, 1933.

Gottlieb, B. (1): Der Epithelansatz am Zahne. Dtsch. Monatsch. f. Zhk. **39**:142, 1921.

Gottlieb, B. (2): Ätiologie und Profülaxe der Zahnkaries. Z. f. Stomat. **19**:129, 1921.

Immenkamp, A.: Ein Beitrag zur Klärung einiger Streitfragen in der keramischen Protetik auf Grund histologischer Untersuchungen. Vjschr. f. Zhk. **48**: 492, 1932.

Wærhaug, J.: The gingival pocket, anatomy, pathology, deepening and elimination. Odontologisk Tidskrift (Odontological Journal), **60**: Supplementum 1, 1952.

Wærhaug, J., and Steen, E.: The presence or absence of bacteria in gingival pockets and the reaction in healthy pockets to certain pure cultures. Odontologisk Tidskrift, **60**:1, 1952.

Case Report

Abrasion Through the Abuse of Dental Floss

by FRANK G. EVERETT, M.S., D.M.D., M.D.,* AND PAUL W. KUNKEL, SR., D.M.D.,†
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GOOD home care of the mouth is accomplished by the patient through the correct use of certain instruments and by proper mouth rinsing.[1] The toothbrush, dental floss and tape, soft and hard wood points and soft rubber cones are some of the tools our patients employ for this purpose[2,3,4,5]; any one of these mechanical aids may occasionally be abused by the patient.[6]

This report deals with a patient, a gentleman 66 years of age, seen in the office of one of us (P.W.K.); this patient, who suffered from advanced periodontal simplex, presented an unusual lesion in the lower front tooth area. Around the lingual and proximal circumference of several of the lower incisors could be noted deep, narrow grooves located slightly apically of the cemento-enamel junction (Fig. 1). On interrogation these lesions turned out to be sequelae of the incorrect and over-zealous use of dental floss.

Fig. 1. Intra-oral roentgenogram of the lower front tooth area. The horizontal dark lines slightly apically of the cemento-enamel junction indicate the grooves worn by the floss. The lesion is most noticeable in the lower right lateral incisor.

Following is the method used by the patient. First he applied toothpaste to the lower front teeth and passed a piece of waxed dental floss about 18 inches long between the incisors; next, he turned the floss around the lingual surface of the tooth and passed it again through the adjoining interproximal space. Then, holding the two ends of the piece of floss in his two hands, he pulled vigorously on one end of the floss, while at the same time approaching the teeth with the other end. The direction of pull was then alternated repeatedly (Fig. 2). The whole procedure somewhat resembled the way in which a bootblack shines the heel of your shoes with a long piece of flannel.

The considerable friction caused by the rapid pulling of this overly long[4] piece of floss around the tooth to which had been applied an abrasive toothpaste had ultimately caused the wearing of deep circular grooves around the teeth thus treated. By use of this incorrect method over many years the patient had sawed his lower incisors almost in half. At least one of them, the lower right lateral incisor, was at the point of breaking off.

This short report may serve to re-emphasize the well-known fact that pa-

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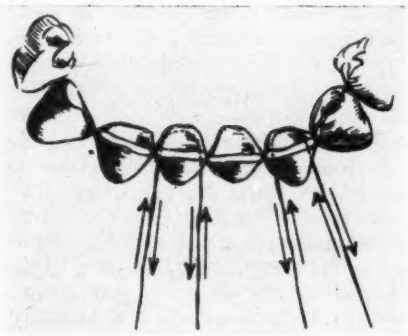

Fig. 2. Diagram showing the incorrect use of dental floss by the patient.

tients have to be taught repeatedly how to employ any of the tools used in home care of the mouth and should be made to demonstrate their skill in subsequent appointments, or else they may misunderstand instructions and cause considerable damage to their teeth or investing tissues.

BIBLIOGRAPHY

1. Everett, F. G., and Bettman, M. M. Mouth Rinsing. J. Periodontology **23**:213-215, 1952.

2. Bell, D. G. Teaching Home Care to the Patient. J. Periodontology **19**:140-143, 1948.

3. Leonard, H. J. Home Technics for the Care of the Mouth. J. Periodontology **20**:27-31, 1949.

4. Hard, D. G. The Conservative Treatment of Periodontal Disease. J.A.D.A. **44**:725-733, 1952.

5. Haverstick, E. E. When and How to Use Dental Floss. Dental Cosmos **75**:1179-1181, 1933.

6. Periodontal Workshop. Report of Evaluating Committee. II. Local Factors in Periodontal Disease. J.A.D.A. **45**:16-20, 1952.

Results of a Three Year Study of Vincent's Infection at the Great Lakes Naval Dental Department

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HISTORY

PROJECT NM 008 013.01, "Factors Involved in Acute Ulceromembranous Stomatitis," was approved by the Chief of the Bureau of Medicine and Surgery, Department of the Navy, on the 15th of April 1947. Naval Training Centers at Great Lakes, Illinois; San Diego, California and Bainbridge, Maryland, were chosen as test centers for this investigation. The various training commands were to collaborate with the dental research sections of the Naval Medical Research Institute and the National Institute of Health at Bethesda, Maryland. It was the mission of the Navy to provide the clinical and laboratory services of the dental officers, medical officers and technicians.

Instructions pertaining to the methods and procedures to be followed were formulated and issued to the three training centers by the end of 1947. This project gave impetus toward setting up a research department in the Dental Department at Great Lakes. Space was allotted and equipment procured under the provisions out-

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The opinions or assertions contained in this article are the private ones of the writer and not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

TABLE A.

Monthly Attack Rates of Vincent's Infection Per 1,000 Personnel During 1951

Month and Year 1951	*Vincent's Cases*	*Unclassified Gingivitis*
January	0.92	1.95
February	1.37	2.36
March	1.48	1.61
April	0.96	2.19
May	1.30	3.34
June	1.14	2.08
July	0.46	0.48
August	1.00	1.38
September	1.65	1.56
October	1.88	2.55
November	2.41	2.70
December	1.66	0.38
Mean for year	1.60	6.41
Median for year	1.46	5.55

lined by the approved project orders. In December of 1948 the study was transferred in toto from the other research centers and training commands to the Dental Department at Great Lakes. The initiation of this study was placed with the Senior Dental Officer who in turn designated a dental officer as research assistant and collaborator.

This report covers a span of three years from 1949 to 1952. It is a compilation of numerous studies by past and present dental research officers.

OBJECTIVE

The objective of this investigation was to determine the epidemiology of acute ulceromembranous stomatitis and such other factors that might lessen the occurrence or hasten the recovery of patients so afflicted. An important issue that was hoped to be solved was whether "Vincent's Infection" was a disease process or the manifestation of a syndrome. Another vital question of immediate concern to a practitioner of military dentistry is whether it can be considered contagious, and should it be treated as a contagion. It is hoped that additional information will be provided in this report though it is not possible to answer these questions in either the affirmative or negative with conviction.

METHOD

The patients incorporated into this study were from the general population at the Great Lakes Naval Training Center. This group consisted of recruits, service school personnel and permanent staff members of the several commands. There were, at the initiation of this project, five dental dispensaries treating only recruit personnel and one dispensary for staff and service school personnel. Examinations were performed by an appointed "sick call" dental officer in each clinic and subsequently reported to the research officer.

A data sheet was provided each "sick call" dental officer for the entry of pertinent patient historical and clinical information. These sheets were specially designed to show the nature of the contact of the patient prior to onset of the disease, and a review of his exposure to both public and private activities. The sheet also provided spaces for a complete history and all associated subjective and objective symptoms. This information, plus a differential diagnosis and record of treatment, was placed in a four page portfolio that could be readily transferred to a punch card for statistical analysis.

After the patient had been examined and interrogated, he was transferred to the research laboratory for a repeat clinical examination and subjected to various laboratory procedures. The hematology tests included a total RBC, WBC, hemoglobin, differential, red and white cell morphology, a Wassermann and Kahn and a Vitamin "C" determination of the blood plasma. Bacteriological examination included a dark field, stained smears and culture of the fluid beneath typical papillae. The pathology procedures consisted of aspirating fluid from the affected interproximal spaces and inoculating this material into the cornea of the eye of a rabbit. Following a prescribed interval, the eyes

were examined for gross pathology, and later (after the animals were sacrificed) for microscopic pathology. This procedure was followed to test for the presence of Herpes Simplex virus. In conjunction with this, gingival biopsies were taken and examined for the tissue changes.

The monthly attack rates were tabulated from the number of reported cases by each clinic and computed in percentage of the total on board compliment for the month (Table A, B, C). It was soon discovered that there was considerable disparity in the diagnosis of "Vincent's Infection," thus many reported cases had to be ruled out. During the first six months, a total of 171 cases were reported but further critical examination pared this number to only 22. Those cases that did not meet the requirement for "Vincent's Disease" were called "unclassified gingivitis," and will appear as such in some of the results.

The clinical diagnosis for the condition called "Vincent's Infection" depended upon the presence of all three of the following symptoms: (1) the presence of ulcerated interdental papillae with necrosis of the tissue, (2) the appearance of a grayish slough covering the ulcerated areas, and (3) the presence of fetid odor. A differential diagnosis was made to exclude the following: scurvy, syphilis, diphtheria, streptococcic sore throat, migrating cheilitis, blood dyscrasias, tuberculosis, diabetes, metallic poisoning, aphthous stomatitis, hypertrophic gingivitis, thrush, suppurative periodontitis and traumatic ulceration.

RESULTS

The first group of cases under study were for the purpose of proving or disproving facts already formulated by previous investigators. The results of this series of experiments were tabulated on a total of 109 cases and are separated into three phases; first, the clinical aspect, second, the laboratory examination of blood and tissue fluids, and third, the pathology of human and animal tissues.

TABLE B.

Monthly Attack Rates of Vincent's Infection Per 1,000 Personnel During 1950

Month and Year 1950	*Vincent's Cases*	*Unclassified Gingivitis*
January	2.44	4.64
February	1.50	4.25
March	1.16	2.91
April	2.41	1.131
May	1.76	2.04
June	1.15	1.51
July	1.22	1.97
August	1.59	2.96
September	1.00	2.94
October	1.47	4.07
November	1.40	1.93
December	0.70	1.86
Mean for year	1.48	2.68
Median for year	1.36	2.30

In this category of clinical subjective and objective symptoms, the condition noted is calculated percentage wise relative to the total group of 109 cases. There are instances where symptoms show only a remote relation to the disease and have thus been excluded from this report. Below are listed the various categories and their percentage occurrence:

1. *Subjective and objective symptoms.*

AGE: The patients ranged in age from 17 to 29 years with an average age of 19 years. Since the largest portion of the population at this base is recruits, it is only logical that the majority of patients would fall in the age of new enlistees.

CHIEF COMPLAINT: Pain and bleeding were the principal reasons for the patient choosing to visit the "sick call" officer. Pain was the causative factor in 44 per cent of the cases, while bleeding was responsible in 25 per cent of the cases. Pain and bleeding both were only reported in 5 per cent of the cases.

NATURE OF CONTACT: Due to the insiduous onset of the disease, none of the cases were able to identify a possible previous carrier. Even the cases that had contact with well patients prior to reporting to the "sick call" officer did not indi-

TABLE C.

Monthly Attack Rates of Vincent's Infection Per 1,000 Personnel During 1949

Month and Year 1949	*Vincent's Cases*	*Unclassified Gingivitis*
January	1.86	3.37
February	1.75	4.54
March	1.28	6.61
April	2.15	5.24
May	1.26	6.98
June	2.24	1.55
July	1.37	7.57
August	0.62	4.80
September	0.84	3.35
October	1.19	7.50
November	2.22	12.14
December	2.53	13.28
Mean for year	1.35	1.88
Median for year	1.27	1.88

cate transmission of the disease. Association with other than naval personnel was quite limited and 97 per cent claimed they were on duty throughout the week prior to reporting their case.

SPECIAL CIRCUMSTANCES: Overindulgence in drinking and smoking was affirmed in 48 per cent of the cases, while petting was acknowledged in only 2 per cent of the cases. The latter does not appear too low in view of the fact that social life is quite restricted for the recruit.

SUBJECTIVE SYMPTOMS: Patients complained of pain in the gingivae by 74 per cent of the total cases, and the pain persisted for a range of 1 to 8 days. Headaches were reported in only 8 per cent of the cases and were not long lasting or very severe. Profuse bleeding was presented in 93 per cent of the cases, lasting for a range of one to twelve days. Malaise was experienced in 16 per cent of the cases, but was never of such intensity that required hospitalization. Better than 95 per cent of the cases showed a slight elevation in temperature of from one to three degrees. Diarrhea was present in 6 per cent of the cases but could not be associated with the severity of the disease.

OBJECTIVE SYMPTOMS: The involved tissue with the necrotic area and sloughing membrane are listed below: (See Table No. 1).

The condition of lymphadenopathy was scored and is listed in the frequency of occurrence and duration. (See Table No. 2).

As was mentioned in the criteria, all cases presented with necrosis of the papillae, pseudomembrane formation and a fetid odor. Erupting third molars were noted in 20 per cent of the cases and malocclusion in three cases. Contributory oral manifestations such as calculus formation (47 per cent of cases), food wedging (10 per cent), and complete dislike for fruit (15 per cent) was also noted.

TABLE No. 1

Involved Tissue With Necrotic Areas and Sloughing Membrane

Tooth Area	*Percent Cases Involved*	*Average Days Involved*	*Range of Day Involved*
Lower incisors	86.2	3.62	1 to 16
Upper incisors	57.8	3.44	1 to 17
Bicuspids	67.8	3.12	1 to 8
Molars	33.0	3.13	1 to 7
Distal L 3rd	19.2	3.29	1 to 8
Tonsilar	.9	4.00	4
Tongue	.9	1.00	1

2. *Hematology and Blood Chemistry:* Five cc. of blood were drawn from each patient for a cell count, differential, hemoglobin, sedimentation rate, Wassermann and a Vitamin "C" determination of the plasma (Method of Abt and Farmer).[1,2]

The hematology studies indicated that all patients were within normal limits for red, white and differential counts. The sedimentation rates and percentage of hemoglobin were also within normal limits. The Wassermann test was negative.

The Vitamin "C" determinations were first done on 100 patients during the Fall of 1949. The mean plasma level was 0.309 mg% with a range of 0.20 to 0.72 mg%. The mean plasma level for 30 normal cases

TABLE NO. 2

The Condition of Lymphadenopathy Listed in Frequency of Occurrence and Duration

Glands Involved	*Percent of Cases*	*Days' Duration*
Parotid	4.0	1 to 12
Submaxillary	25.0	1 to 6
Ant. Cervical	.9	2
Superficial Cervical	.92	2
Posterior Auricular	.92	1

was 0.385 mg%. Similar determinations on 23 normals during the Fall and Winter of 1951-52 showed a mean Vitamin "C" level of 0.91 mg%. A survey of 22 Vincent cases during this same period showed a mean level of 0.602 mg%.

3. *Bacteriological Results:* A study of dark fields showed the *Borrelia vincenti* (loosely coiled organisms with tapered ends), *Borrelia buccale* (double contour organisms), and small oral troponemes (closely spaced spirals, long tapered ends) were usually present.[4,5] The results provided from examining more than two hundred dark fields gives evidence that *Borrelia buccale* was predominant over *Borrelia vincenti* by 80 per cent.

Smears plated on blood agar and nutrient agar slants failed to reveal any consistent findings. The blood plates usually had varying amounts of growth in which the responsible organism was Streptococcus viridans and an occasional hemolytic organism.

Routine smears made from the active site showed no correlation with smears made from different areas in the same mouth when stained with Gram's stain. The appearance of individual spirochetes, fusiform bacilli and cocci varied so greatly in each smear that their predominance could not be determined with any degree of assurance. The fusiform bacilli were usually observed to be Gram negative, but occasionally they would appear as Gram positive. It was further noted that the identification of the spirochetes in stained smears was practically impossible due to the distortion produced by the chemicals in the stain.

Lactobacillus counts were made by plating dilutions of saliva on tomato juice agar (Hadley[6]), (Jay[7]), on all patients but it could not be demonstrated that there was any correlation with the resultant count and the degree or course of the infection.

4. *Pathological Studies:* Biopsies of the infected papillae were taken on the more severe and highly typical cases, and this tissue processed, stained and sectioned. There was one common finding of all the sections, the fact that keratinization was lacking. The histopathology of the underlying tissue was one of an inflammatory reaction and areas of necrosis. The removal of gingival tissue for the biopsies did not appear to affect the course of the infection, in fact, there was evidence that these resected areas healed faster than the undisturbed areas. This phonomena was probably due to the elimination of a focal site for bacterial propagation.

Inoculum was taken from infected gingival crevices and injected into the scarred surface of the cornea of the eye of an anesthetized rabbit. When the cornea showed a gross pathological change, the rabbits were sacrificed and the eyes were then processed, fixed, stained and sectioned for microscopic examination. Herpetic vesicles which would indicate the presence of a virus were not observed.

5. *Epidemiology Studies:* Studies in this respect fail to shed any light on possible epidemic tendencies of this disease. New cases failed to appear from barracks in which infected men were berthed, and there was no evidence that any of the cases were a direct result of any specific mess hall contaminant. The cases observed were fairly evenly distributed within the confines of the Training Center. It did appear that there were more new cases following a station wide holiday that involved the absence of personnel from the Center for a week or more. A review of the individual case histories denoted a lack of evidence that smoking, drinking or possibly contamination from petting provided factors in establishing the etiology or epidemiology

of the disease. Neglected oral hygiene could be a contributory one. Another contributory factor could be explained from the lowered resistance brought about by environmental changes and the participation in stimulating activities during vacation periods. (See Tables A, B and C.)

RESULTS

Epidemiological studies were conducted at this Center for the purpose of determining the communicability of ulceromembranous stomatitis. Nine weekly surveys were made on 70 men berthed in the same dormitory and fed in the same galleys. The survey consisted of assessing the gingival tissues by the same diagnostician after the method of Massler, Schour and Chopra.[8] The number of affected gingival units were recorded for each man weekly.

During the fourth week of the survey period, one member of the group was diagnosed as having ulceromembranous stomatitis. This man was not informed of his condition and was not treated. By the fifth week the ulceromembranous stomatitis had resolved into a subclinical gingivitis and remained unchanged during the remaining weekly surveys. There was no indication from the recordings of the other 69 men that transmission had occurred.

Therapeutic efficacy was determined by the use of several chemical and antibiotic compounds. All cases were classified according to the clinical degree of severity, being categorized into mild, moderate and severe. The condition was treated for the first 48 hours by the medicament under investigation and then followed by scaling, toothbrush technique and other aids. The criterion for determining the beneficial effects of each medicament was the rapidity in which the patient's gingivae returned to normalcy. Other conditions noted as the patient progressed, were the loss of soreness, resolution of the grayish membrane and the return of the tissues to normal color and tone. Below are listed the medicaments used, degree of severity and length of treatment.

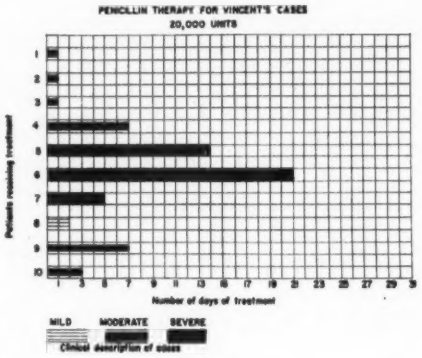

Fig. 1.

CLINICAL ASSAY OF TREATMENTS INSTITUTED

The first group of patients treated (Fig. No. 1) received a lime flavored Penicillin-Bacitracin troche containing 20,000 units of Penicillin and 50 units of Bacitracin. The treatment schedule consisted of one troche every two hours for two days or until a total of 20 troches had been taken. Whatever additional treatment was deemed necessary began on the third day by the clinician.

The mean number of days necessary to return patient's gingivae to normal was 6.2. Patients found this troche agreeable to take and most of them enjoyed the taste.

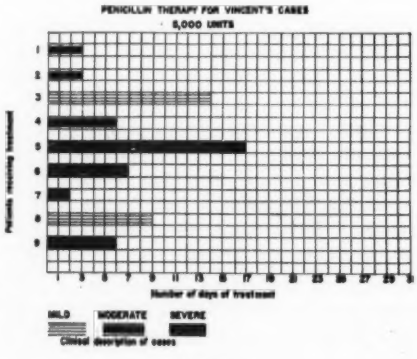

Fig. 2.

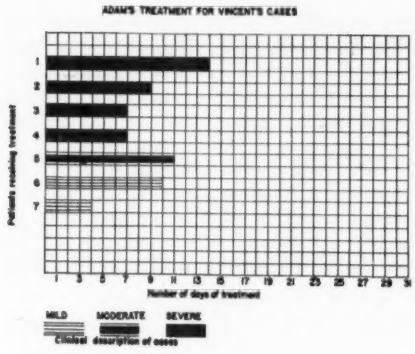

Fig. 3.

Pain associated with the condition was greatly improved by the end of the first day and was completely absent by the end of the second day of treatment. The grayish membrane was usually absent by the end of the second day.

The advantages of this type of treatment are that results are quickly obtained, troches are easily administered, patient time is greatly reduced and treatment is pleasant to the patient. The disadvantages to this form of treatment are: possible side reactions responsible to the active ingredients of the troches; no assurance that patient will adhere to instruction for self medication and psychological effects of impersonal office care.

The second group study (Fig. No. 2) received a peppermint flavored Penicillin troche containing 5,000 units of Penicillin and a fungicide (Benzyl Alcohol, 0.3%, and Cetylpyridium Chloride, 1:1500). The treatment schedule consisted of one troche every two hours for two days or until a total of 24 troches had been taken.

The mean number of days necessary to return the patient's gingivae to normal was 7.0. Patients reported a dislike for this troche due to the flavor and also to the number of troches prescribed. Pain was only moderately improved by the end of the first and second day of treatment. The grayish membrane was slower in resolving in comparison to the response to the 20,000 unit Penicillin troche.

The advantage of these troches was similar to that of the 20,000 unit Penicillin troche except that the gingivae showed only moderate improvement by the end of two days treatment.

Adam's treatment (Fig. No. 3) was provided for in the office by the dental officer. The procedure consists of isolating one quadrant of the mouth with cotton rolls; application of Churchill's Iodine to the gingival margin and interproximal spaces followed by immediate flushing of the treated areas with 10 per cent silver nitrate and then rinsed thoroughly with warm water. The mean number of days necessary to return the gingival tissue to normal was 9.6. The patients experienced immediate relief upon completion of the first treatment. By the end of the second day, little discomfort was noted. The grayish membrane was usually resolved by the end of the two days of treatment. Patients found this form of treatment provided quick relief for the symptoms of pain and bleeding. The patient-dental officer relationship in which personal attention was given was preferred by the majority of patients. The disadvantages in this form of treatment are: time involved and precaution in use of medicaments.

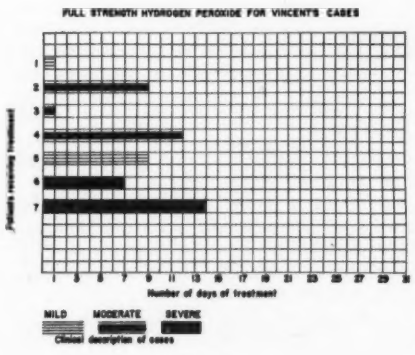

Fig. 4.

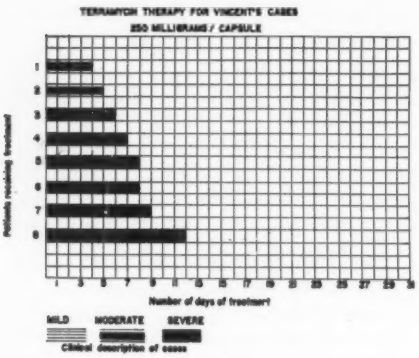

Fig. 5.

The fourth group receiving therapy (Fig. No. 4) were given 3 per cent Hydrogen Peroxide in the form of an aerosol and oral rinse. The patient upon admission with the infection, had his mouth thoroughly sprayed with 3 per cent Hydrogen Peroxide and then given a 60 ounce bottle of 3 per cent Hydrogen Peroxide to be used at home following each brushing of the teeth.

The mean number of days necessary to return the gingivae to normal was 7.5.

The symptoms of pain and bleeding were not relieved by the end of the first day of treatment and seldom by the end of two days of treatment. The grayish membrane was only moderately resolved by the end of two days of treatment.

The advantages to this form of treatment are similar to those of Adam's treatment with the exception that relief was not as perceptible except in mild cases. The fetor oris associated with the disease is reduced considerably. Disadvantages of this form of treatment are: patients dislike medicament; difficulty in having patient follow instructions and inability to obtain improvement of the condition except in mild cases.

The final preparation used in therapy for ulceromembranous gingivitis was Terramycin (Fig. No. 5). Capsules containing 250 mg. each were given to the patient with instructions to take two capsules every six hours until a total of 2 gms per day had been taken.

The mean number of days necessary to return the gingival tissue to normal was 7.3. The majority of cases treated were severe in type and showed outstanding relief from pain by the end of the first day. The grayish membrane was almost completely resolved after one day of treatment.

The advantages for this type of treatment are similar to those offered in the discussion on Penicillin troches, but appear clinically to be more rapidly effective. The disadvantages include cost of preparation, inability to make certain patients follow directions, possible deleterious side effects and possible interruption of therapy by the fact that quick relief is afforded.

BIBLIOGRAPHY

1. Farmer, C. J., and Abt, A. F. Ascorbic Acid Content of Blood. Proc. Soc. Exp. Biol. and Med. **52**:1625, June 1935.

2. Farmer, D. J., and Abt, A. F. Determination of Reduced Ascorbic Acid in Small Amounts of Blood. Proc. Exp. Biol and Med. **34**:146, March 1936.

3. Linghorne, W. J., McIntosh, W. G., Tice, J. W., Tisdall, F. F., McCreary, J. F., Drake, T. G. H., Greaves, A. V., and Johnstone, W. M. Relation of Ascorbic Acid Intake to Gingivitis. J. Canadian Dent. Assoc., **12**:49-66, Feb. 1946.

4. Hampp, E. G., Scott, D. B., and Wycokoff, R. W. G. Morphologic Characteristics of Certain Cultured Strains of Oral Spirochetes and Treponema Pallidum as Revealed by Electron Microscope. Journal of Bacteriology **56**:755-769, December 1948.

5. Hampp, E. G. Comparative Study of Dark Field and Stained Smear Techniques for Identification of Oral Spirochetes on the Basis of Morphologic Characteristics. J. Am. Dent. Assoc. **32**: 318-324, March 1945.

6. Hadley, F. P. Quantitative Method for Estimating Bacillus Acidophilus in Saliva. J. Dent. Res., **13**:15, Oct. 1933.

7. Jay, P. J. Nutrition and Caries. New Jersey Dent. Soc. **15**:20-24, 1944.

8. Massler, M., Schour, I., Chopra, B. Occurrence of Gingivitis in Suburban Chicago Children. J. Periodontology, **21**:146, 1950.

Announcements

PRELIMINARY PROGRAM

American Academy of Periodontology
Cleveland, Ohio

Thursday—September 24, 1953

A.M.

11:00-11:40 The Normal Physiology of the Periodontium as it is Affected by Occlusal Function.
—Charles Williams

11:40-11:50 Discussor—John F. Prichard

11:50-12:00 General Discussion

Luncheon

P.M.

2:00- 2:20 The Blood Supply of the Gingival Tissue—Walter Cohen

2:20- 2:30 Discussor—B. O. A. Thomas

2:30- 2:50 The Reattachment of Human Periodontal Tissues Following Surgical Detachment.
—Melvin L. Morris

2:50- 3:00 Discussor—Sigurd Ramjford

3:00- 3:20 Alveolar Bone Changes Following Alteration of Masticatory Function in the Rat.
—Sidney L. Horowitz and Harry H. Shapiro

3:20- 3:30 Discussor—Joseph P. Weinman

3:30- 3:50 The Effect of Trauma to the Periodontal Membrane on the Spread of Gingival Inflammation.
—Joseph P. Weinmann and Luz Macapan

3:50- 4:00 General Discussion

4:00- 4:40 Treatment Planning of Deep Overbite Cases.
—Herbert I. Margolis

4:40- 4:50 Discussor—Lewis Fox

4:50- 5:00 General Discussion

Friday—September 25, 1953

A.M.

9:00- 9:40 Protein Deficiencies in Animals and Effect on Attachment Apparatus.
—Henry M. Goldman

9:40- 9:50 Discussor—Paul Boyle

9:50-10:00 General Discussion

10:00-12:00 Symposium on Oral Lesions
- 10:00-10:30 Clinician—David Weisberger
- 10:30-11:00 Radiologist—Harry Hauser
- 11:00-11:30 Pathologist—Robert Gorlin
- 11:30-12:00 General Discussion

P. M.

12:30- 2:15 Round Table Luncheon—Operation for Coronary Artery Disease (Motion Picture)—Claude S. Beck

2:30- 4:00 Symposium on Bone—Bone Resorption
- 2:30- 3:10 (a) Biochemical and Biomechanical Aspects of Bone Resorption—Franklin C. McLean
- 3:10- 3:40 (b) Periodontal Aspects—Balint Orban
- 3:40- 4:00 General Discussion

4:00- 5:15 Panel—Undergraduate & Graduate Training Related to Certification
- 4:00- 4:10 "Undergraduate Instruction in Periodontology" —B. O. A. Thomas
- 4:10- 4:20 "Graduate Instruction in Periodontology" —H. M. Goldman
- 4:20- 4:30 "Minimum Requirements for Certification" —Harold J. Leonard
- 4:30- 4:40 "Minimum Requirements for Certification" —James R. Blayney
- 4:40- 5:00 Exchange of questions between the experts on the panel.
- 5:00- 5:15 General Discussion

7:30 ANNUAL BANQUET

Saturday—September 26, 1953

A.M.

9:00-11:00 Round Table Discussion on Various Surgical Treatments of Marginal Gingivitis and Periodontitis.
- 9:00- 9:15 Curettage—Bernard S. Chaikin
- 9:15- 9:30 Gingivoplasty—Robert Gottsegen
- 9:30- 9:45 Gingivectomy & Interdental Resection—Richard Chace
- 9:45-10:00 Osseous Resection—Saul Schluger
- 10:00-10:15 Flap Approach—Jack L. Bailin
- 10:15-10:30 Exchange of questions between experts on panel
- 10:30-11:00 General Discussion

Saturday afternoon—Clinic Program

Dr. E. Bruce Clark, Pittsburgh, Pennsylvania
"The Use of a New Type Hinge-Axis Locator in Mounting Study Casts"

Dr. Jerome J. Hiniker, Falls Church, Virginia
"Gingivectomy and Electrosurgery"

Dr. W. G. F. Schmidt, Chicago, Illinois
"The Correction of Occlusion of Natural Teeth as an Aid in Treatment of Periodontia"

Dr. Frank Wentz, Loyola School of Dentistry, Chicago, Illinois
"Clinical Pathology of the Oral Mucous Membrane"

Dr. John Ingle, University of Washington, School of Dentistry, Seattle 5, Washington
"Esthetics and Surgery in the Premaxilla"

BETH ISRAEL HOSPITAL

The Beth Israel Hospital, Boston, Massachusetts, will offer a postgraduate course in Periodontia consisting of lectures, seminars, and clinical work. The course is conducted under the supervision of Drs. Henry Goldman, Bernard Chaikin, and Lewis Fox. The dates are October 5 to 16, 1953.

Those wishing to apply may contact the Director of the Dental Department at the Beth Israel Hospital.

PERIODONTOLOGY COURSES AT COLUMBIA UNIVERSITY

Four postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

A full-time program covering a period of two academic years and a half-time program covering a period of four academic years lead to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

Of the two part-time courses, one course will be given one full day a week from September 3, 1953 to May 5, 1954; the second course will be given one half day each week from September 23, 1953 to May 5, 1954.

For further information write to Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th St., New York 32, N. Y.

UNIVERSITY OF MINNESOTA

The University of Minnesota announces the Eighth Annual Oral Medicine Seminar, to take place September 17, 18, 19, 1953 at the Center for Continuation Study.

September 17 Symposium: "Anatomy and Growth of the Head and Jaws: Surgical Correction of Abnormalities"

Harry H. Shapiro, Assistant Professor Anatomy, Columbia University.

Wilton M. Krogman, Professor of Physical Anthropology, University of Pennsylvania

Bernard G. Sarnat, Professor of Oral and Maxillo-facial Surgery, University of Illinois

September 18 Symposium: "Finding of Recent Research Related to Operative Dentistry, and Application to Practice"

Sumter S. Arnim, Professor of Pathology, University of Texas

Joseph F. Volker, Dean School of Dentistry, University of Alabama

Ralph W. Phillips, Associate Professor of Dental Materials, University of Indiana

September 19: S. W. Leung, Professor of Physiology, University of Pittsburgh, "The Effect of Saliva on the Oral Tissues."

S. J. Kreshover, Professor of Oral Pathology, Medical College of Virginia, "Pre-natal Influences and Tooth Development."

Further information may be obtained by writing to the Director, Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.

EXAMINATION BY AMERICAN ACADEMY OF ORAL PATHOLOGY

The American Academy of Oral Pathology will conduct its examination for the elevation of members to fellowship on Friday, the 16th of October, 1953. The examination will be held at the Armed Forces Institute of Pathology, Seventh Street and Independence Avenue, Southwest, Washington, D. C., and will begin promptly at 8 A.M. Members of the Academy wishing to apply for permission to be examined for fellowship, should contact Colonel Joseph L. Bernier, D. C., Armed Forces Institute of Pathology, Secretary of the Academy.

TUFTS COLLEGE DENTAL SCHOOL

Tufts College Dental School announces its Graduate and Postgraduate program in Periodontology for the academic year of 1953-54. All courses are conducted by Professor Irving Glickman and staff.

PERIODONTOLOGY I

A full-time two-year specialist training program for individuals interested in qualifying for certification in Periodontology. This program has been arranged to conform with the requirements of the American Board of Periodontology. One-half of this course is devoted to didactic instruction and the remainder to clinical practice.

PERIODONTOLOGY II

A one-year full-time specialist training program, this is a modification of the two-year course and is offered to individuals who have had a year's previous graduate or postgraduate training in Periodontology.

PERIODONTOLOGY III

An 18-month full-time course leading to an M.S. or M.S.D. Degree. This course consists of didactic instruction, pre-clinical sciences, clinical training in Periodontology

and research and thesis. The facilities of the various departments and hospitals of the medical and dental schools are available for research activities.

DPG. 601-PARTICIPATION COURSE IN PERIODONTOLOGY

Two-weeks—October 5-17, 1953

Class limited to 12 Tuition $250.00

Half of the time of this course is devoted to clinical practice by the participants. The remainder of the curriculum consists of lectures and seminars devoted to the theoretical and didactic aspects of periodontology. The histopathology of periodontal disease and the "Bone Factor" concept will be presented. The clinical management of periodontal disease is analyzed in detail with special consideration given to the various aspects of diagnosis, determination of prognosis and treatment planning.

DPG. 602-PARTICIPATION COURSE IN PERIODONTOLOGY

April 12-16, 1954

Class limited to 12 Tuition $150.00

This one week course consists of lectures and clinical sessions devoted to the various aspects of periodontal disease and their treatment. Half of the time is devoted to clinical practice by the participants under the direction of Dr. Glickman.

DPG. 603-KIRKLAND GINGIVECTOMY TECHNIQUE

May 17-21, 1954

Class limited to 12 Tuition $125.00

In this course the concept and technique for the Kirkland Gingivectomy operation is presented. Each step in the technique is discussed and demonstrated. The participants operate in the clinic under the direction of Dr. Olin Kirkland.

DPG. 604-OCCLUSAL EQUILIBRATION

Each Monday for five weeks

Jan. 11-Feb. 8, 1954

Class limited to 12 Tuition $150.00

Dr. Edward T. Fisher

This course is devoted to the principles of Occlusal Equilibration as they apply to the treatment of periodontal problems. It will consist of lectures, exercises on models, clinical demonstrations, and participation by students. Diagnosis and case management will be stressed.

For further information and application, write to Director Graduate and Postgraduate Studies, Tufts College Dental School, 136 Harrison Avenue, Boston, Massachusetts.

WINS TUFTS BERKSHIRE CITATION

Dr. Arthur H. Merritt, of New York City, recently received the Tufts College Dental School "Award for Leadership in Periodontology" at the fourth annual Berkshire Conference held in Lenox, Mass.

AMERICAN INSTITUTE OF DENTAL MEDICINE

Between three and four hundred dentists, including top dental and medical researchers, will be in attendance at the 10th Annual Meeting of the American Institute of Dental Medicine, at the Desert Inn and El Mirador, Palm Springs, California, October 25 through October 29, 1953. Advance registration, according to an announcement by Dr. Hermann Becks, Director and Founder of the Institute, indicates a capacity turn-out for the five-day study session. Included in the lecture-packed program to be discussed and analyzed at the meeting are the following subjects:

W. L. Bostick, M.D., University of California, San Francisco, California: Dynamics of Inflammation; Cellular Reaction to Injury and The Malignant Lymphoma.

A. C. Curtis, M.D., University of Michigan, Ann Arbor, Michigan: The Physiology of Pigmentation in Man; Cutaneous Virus Diseases; The Dentist Looks at the Field of Dermatology.

H. B. G. Robinson, D.D.S., University of Ohio, Columbus, Ohio: The Oral Pathologist Considers Focal Infection; Clinical Aspects of Intraoral Ageing and Recalcitrant Diseases of the Oral Mucosa.

D. B. Scott, D.D.S., National Institutes of Health, Bethesda, Maryland: The Structure of Normal Dental Tissues as Observed Under the Electron Microscope; Electron Microscopy of Carious Enamel and Dentin; The Influence of Structural Variation on Current Histological Concepts.

H. Selye, M.D., Ph.D., University of Montreal, Montreal, Canada: The Alarm Reaction; The General Adaptation Syndrome, and The Role of Stress and of the Adaptive Hormones in Dental Medicine.

H. Sicher, M.D., Loyola University School of Dentistry, Chicago, Illinois: Anatomic Basis for the Spread of Dental Infections; Problems of Pain in Dentistry and Common Misconceptions in Genetics.

These and other subjects along the same lines will be discussed and all Seminar lecturers will participate in a round table forum covering the application of the subjects to the practice of Dental Medicine. Applications and full information on the Seminar program may be secured by writing Miss Marion G. Lewis, Executive Secretary, 2240 Channing Way, Berkeley 4, California.

(Continued from page 155)

22. Weinmann, Joseph. Stoffweckselbefunde bei der diffusen Atrophie des Alveolarknockens. Ztschr. f. Stomatol. **25**:1, 1927.

23. Schour, Isaac. The Effect of Hypophysectomy in the Periodontal Tissues. Jour. Periodont. **5**: 15, 1934.

24. Ray, Harold G. A Study of the Histopathology of the Gingiva in Patients with Diabetes Mellitus. Jour. Periodont. **19**:128, 1948.

25. Goldman, Henry M. Periodontia. St. Louis, The C. V. Mosby Co., 1942. p. 171.

26. Talbot, Eugene. Interstitial Gingivitis and Pyorrhea Alveolaris. The Ranson and Randolph Co., Toledo, Ohio. 1913, p. 97.

27. Boenheim, Felix. Endocrine Therapy of Periodontosis. Jour. Dent. Res. **17**:107, 1938.

Idem: Pathologic Importance of the Endocrine Glands in Parodontal Disease. Jour. Dent. Res. **17**:19, 1938.

28. Hirschfeld, Isador. Treatment of Suppurative Periodontitis—Conservative Viewpoint. N. Y. Jour. Dent. **18**:84, 1948.

29. Gottlieb, B., and Orban, B. Biology and Pathology of the Tooth and Its Supporting Mechanism. Translation by M. Diamond, N. Y. Macmillan Co., 1938, p. 73.